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BUREAU OF HYGIENE AND TROPICAL DISEASES.

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MEDICAL ORGANIZATION AND DISEASES OF THE NETHERLANDS EAST INDIES

BEFORE THE JAPANESE INVASION

By CHARLES WILCOCKS, M.D., M.R.C.P., D.T.M. & H.

Acting Director, Bureau of Hygiene and Tropical Diseases.

The total population of the Netherlands East Indies was 60,727,000 in 1930, and by 1938 had, it was estimated, increased by about 4,000,000. In Java and Madura alone there are 42,000,000 inhabitants, concentrated to a density of 314·5 per square kilometre. In other and larger islands the population is much less dense, but Bali and Lombok are relatively closely populated.

ORGANIZATION.

The medical services date from the time of Bontius (1592–1631). There is now a large public health service which works in active cooperation with the military medical service, the missions and local administrative bodies. There are also many private practitioners in the towns and on the big estates. It is the view of the Government that curative medicine belongs properly to the sphere of private enterprise and of the local administrative bodies.

By 1936 there existed the following medical institutions:—500 hospitals of various sizes, 15 asylums for the insane, 42 leper colonies, 6 sanatoria for tuberculosis (since increased to 10) and 970 dispensaries. Many of these are administered directly by the Government Medical Service, many by outside bodies with subsidy from Government, and many by purely private enterprises.

For the training of medical men there are two Medical Schools, one at Batavia and one at Sourabaya, and there is a school for dentists. At these institutions natives graduate—in 1936 there were 363 medical students, including 16 women—the graduates practising in the Netherlands Indies and forming the greater part of the medical services there.

The auxiliary staffs of the medical department are trained at many places. There are 18 categories in all, from nurses of many grades to midwives, malaria and plague assistants, vaccinators, laboratory technicians and subordinate employees.

In the public health service and that of the native States there were, in 1939, 170 physicians, 325 native doctors, 16 other medical officers and 24 semi-official doctors. In private practice there were 526 physicians and 163 native doctors. In addition there were 163 military medical officers, of whom 52 worked with the public health authorities. Dentists in private practice numbered 157. There were 82 European and 1,209 native nurses and 225 midwives in official employ, with 476 private midwives, and 426 vaccinators, 55 technicians and 12 pharmacists, while 372 pharmacists were privately employed.

For the large population of these islands this medical organization is still far from adequate, but as time goes on the service will increase as more natives graduate from the medical schools.

Hospitals fall into three classes :—those controlled by Government, those administered by private bodies with Government subsidy, and those controlled solely by private enterprise. The three largest Government hospitals are at Batavia (1,000 beds), Samarang (1,000 beds) and Sourabaya (643 beds). Of the subsidized hospitals the Petronella hospital, in the Djokja district, is one of the largest ; it is surrounded by daughter establishments which are in close touch with it by telephone and travelling clinic.

Under the direction of the public health service are the Pasteur Institute and Government smallpox institute, Bandoeng, at which all kinds of vaccines are prepared. The medical laboratory at Batavia is also the headquarters of the malaria control organization, and there is a laboratory at Bandoeng which deals especially with problems of water analysis. There are four quarantine stations.

Maternity and Child Welfare centres are established at several places ; the number of confinements supervised by midwives is steadily increasing and in 1939 amounted to 22,720, while in Batavia almost two-thirds of the deliveries are conducted by these trained women. There are 76 pharmacies under the care of trained men.

In spite of the long association of the natives with the Dutch, they still retain a large measure of distrust of western medicine and a deep faith in their own healers. To counteract this distrust, instruction in the elements of hygiene is given in schools, but there is also a bureau for popular health literature, which has produced booklets on hygiene and pamphlets on hookworm, cholera, eye diseases, scurvy, rabies, beriberi, maternity and child welfare, and other subjects. Many of these have been sold, and others are available in the People's Libraries.

Insect-borne Diseases.

Malaria.—As in all tropical countries, malaria is the commonest disease and constitutes the most important public health problem.

Before passing on to the distribution of malaria and its relation to the species of *Anopheles* indigenous to these islands, it is well to consider the general position. Malaria causes more deaths than any other disease, and is so widespread that there can be few persons, except young children, living between sea level and the highlands at 5,000 feet who are not affected at some time. In the endemic areas the disease causes high infant mortality, though the birth rate is not particularly low, and in these areas the general death rate at all ages is higher than in those places where malaria is less prevalent. The general death rate is about 20 per 1,000 per annum ; the rate in the endemic areas (where the disease is rife) may rise to 25–50 ; it reached 400 in the

Cheribon Regency during an epidemic in 1917. It is stated that the malaria death rate is higher in towns than in the rural areas.

In the Dutch East Indies malaria is particularly intense in the coastal districts, where it is transmitted by *Anopheles sundaicus* (*A. ludlowi*), a species which breeds in brackish pools open to the sun, for instance where mangrove growth has been cut, and in lagoons and brackish fishponds. This mosquito breeds in association with certain algae, which float on the surface of the fishponds. A fresh water form is found in the interior of N. Sumatra. *A. sundaicus* is always dangerous ; it attacks man readily, and in a series of examinations of over 20,000 specimens a relatively high proportion have been found infected. *A. sundaicus* is not found in Celebes, where the brackish water breeder is the relatively harmless *A. subpictus* (*A. rossi*). Coastal malaria is also transmitted in some areas by *A. umbrosus*, which is also found in the interior of Borneo and in the island of Banka.

In the inland plains and mountains malaria is transmitted by *A. aconitus*, a species which breeds in rice fields, fresh water fish ponds, canals and occasionally in streams. In rice fields the breeding is largely seasonal, taking place after the cutting of the rice, when the fields cannot run sufficiently dry to prevent it. Improvement in the drainage of such fields, has, however, reduced this seasonal breeding to such an extent that not only has the general death rate been lowered, but the natives, who, because of the unhealthiness of the districts, had begun to leave, have returned. In the mountains the important mosquitoes are *A. minimus*, which breeds in the grassy waters of streams, and *A. maculatus*, which is found in streams left open to the sun after the felling of jungle trees. In this respect the malaria caused by *A. maculatus* is man-made and must be taken into account in the agricultural development of the country ; it may be prevented by leaving the stream shade intact.

In Celebes some malaria is transmitted by *A. subpictus*, which may breed in the same manner as *A. sundaicus*, though it is not so dependent on surface algae, but which may also breed in fresh water.

These are the principal vectors of malaria in these islands, but there are others which, though less important, still play a part in transmission. Of these *A. hyrcanus* breeds in the swamps of S. Sumatra and W. Java, in canals and rice fields, *A. kochi* in pools, streams and rice fields, and *A. leucosphyrus* in shaded pools.

The mosquitoes so far referred to are found in Sumatra, Java and Celebes, and the smaller islands near, but in the eastern part of the archipelago the mosquito fauna is quite different. Here, in New Guinea and the surrounding islands, the vectors are :—*A. punctulatus*, which breeds in almost any collection of water open to the sun, such as drains, tins, boats, footprints, clear or turbid water, but not in streams ; *A. punctulatus* var. *moluccensis* which breeds in similar waters and also in large rivers ; *A. barbirostris bancrofti* which in the interior of New Guinea breeds in shade along the banks of small lakes.

It is therefore evident that malaria can be transmitted widely throughout the Dutch East Indies, but, as has been stated, the distribution is not uniform. On the coast it is general and constant, inland the incidence depends on factors such as the clearing of hill streams, the cultivation of rice, or the presence of swamps or hill streams ; it may, therefore, show seasonal variations of great importance, and unusual conditions of rainfall may, by creating breeding places in abundance, so increase the number of mosquitoes that malaria may be spread in

epidemic form. This is particularly the case in those areas where transmission is not normally equal all the year round, and where the state of immunity of the population is not high.

The three common forms of malaria, benign tertian, malignant tertian and quartan, are found throughout. It is the malignant tertian which is the most serious and which causes the highest mortality. Blackwater fever, a sequel of malignant tertian, is found sporadically.

Control measures vary with the habits of the species concerned. For *A. sundaicus*, breeding in salt water fish ponds, very efficient control is obtained by draining once each month. This kills the surface algae on which the mosquito larvae depend. To avoid destroying the fish (which are bred for food) ditches are dug round the ponds, into which the fish may retreat during the process of drying of the fish pond proper. Fresh water fish ponds are either dried out and converted into rice fields, or are stocked with small fish which prey upon the mosquito larvae. In the rice fields breeding takes place shortly after the cutting of the rice. For control the fields are planted all at the same time, and after reaping are completely dried. In the earlier stages of flooding the rice fields are allowed to remain wet for nine days and then dried for two, the cycle being repeated. Irrigation water is carefully controlled, and the channels so constructed that sluggish backwaters are avoided.

In the hills where *A. maculatus* may breed, control is effected by maintaining the shade of natural vegetation along the streams.

In recent years there has been an increase in malaria in those areas in which the house improvement campaign for the suppression of rats (and therefore of plague, see below) has been pressed. The reason for this is that in the process of house improvement breeding places are created, small pools and puddles where earth has been taken, unless great care is exercised. Much of the house improvement is done by the natives themselves, and supervision from the point of view of malaria is not easy.

Plague.—In the Dutch East Indies plague has been present since the great eastern pandemic of 1894, but from Sumatra and Celebes it soon disappeared. In Java it still persists and has risen and fallen in great epidemic waves, the last three of which occurred in 1913–15, 1920–27 and 1932–36. In the inter-epidemic periods, moreover, considerable numbers of cases have been reported. In the earlier years the epidemic foci were in central and east Java, in the later years in west Java. The known deaths from plague during the period 1911–36 numbered 207,666.

In Java, human plague is almost exclusively a disease of the natives, whose houses are infested with rats. It is found predominantly in the hill country above 1,600 feet, especially at the beginning of the rainy season in December and January. Bubonic plague is, as in other countries, the commonest form. In Java the case mortality from this form is very high, usually from 60 to 70 per cent. Plague pneumonia may arise in the course of bubonic plague ; the sputum is laden with bacilli and these are expelled into the air by the patients in the acts of coughing or even talking. There is great danger that persons in contact with these patients will contract the disease through the inhalation of droplets of sputum expelled by the patients. Pneumonic plague is invariably fatal ; it is found in Java in small outbreaks of 2 to 10 cases, and accounts for 6–8 per cent. of the total plague cases seen.

The rats affected are the brown Malayan house rat *Rattus rattus diardii*, *Rattus concolor* inland, and, to a very small extent *Rattus*

norvegicus, the brown sewer rat. The principal flea involved is the tropical rat flea *Xenopsylla cheopis*, notorious in this respect throughout the tropics.

In Java control measures fall into two categories: measures of rat control, and protective inoculation. Rat control in ports follows the usual procedure: fumigation of ships, quarantine, and construction of rat proof godowns and stores. In the country districts, where the majority of cases are found, the Dutch have instituted the campaign of house improvement which has as its object the alteration of existing houses, or the construction of new houses, in such a manner that facilities for rat nesting are eliminated. To this end double walls are abolished, so that rats may not nest in the space, bamboos used in building are sealed at each end for the same purpose, or replaced by wooden beams, the use of tiles to replace thatch is encouraged, and regular inspections are made. The Dutch have carried out this campaign without compulsion, but with the aid of a small bonus for each completed house, and report astonishing success; by the end of 1938 no less than 1,525,364 houses had been improved. The unexpected effect of this activity in causing an increase in malaria has been referred to above.

Preventive inoculation has been developed in an unusual manner. The Dutch bacteriologist OTTEN some years ago isolated a bacillus, presumably a plague bacillus, of such feeble virulence that it could not, even in the living state, provoke the disease in small animals. It was found that this organism similarly failed to produce ill effects when injected in man, and that the protective power against plague developed in animals was greater after injection of suspensions of these living bacilli than after the injection of killed vaccines. This method of inoculation with the living vaccine has been applied in Java on a massive scale since 1934, and by 1938 over 7,000,000 persons had been so immunized. The Dutch have great faith in this measure, and certainly, since it was instituted, the epidemic wave of plague has steadily and steeply declined.

The *typhus group of fevers* is represented in the islands by *mite fever* (tsutsugamushi disease), in which the causative organism is transmitted from rodents to man by the larvae of scrub mites, and by *endemic* or *murine typhus*, a disease of rats spread to man by rat fleas. Mite fever, now regarded as identical with the scrub typhus of Malaya and Japanese river fever, is commonly contracted in scrub country, and may particularly be expected in groups of labourers engaged in clearing bush; it is seen especially in northern Sumatra and New Guinea, though a few cases have been reported from Java and W. Borneo. The incidence is not high in peace time, about 500 cases are reported each year from N. Sumatra. The mite incriminated is the larva of *Trombicula deliensis*, but it has been surmised, but not proved, that ticks of the genus *Amblyomma* may also transmit the disease.

Murine typhus is seen in Java. It is not so fatal to rats as plague, and there is not, therefore, the same tendency for fleas to seek human hosts. It is for this reason unlikely that murine typhus would cause extensive outbreaks in man, though in Shanghai and Mexico this form has been transmitted to man and has then spread in epidemic fashion from man to man through the human louse.

Dengue fever is stated to be generally distributed through the islands. It is transmitted by the mosquito *Aëdes aegypti*. This mosquito, in

Africa and South America, is the transmitter of yellow fever, and though yellow fever has never been found in the Far East, the probability of its wide spread, should it be allowed to enter, is not in doubt.

Infant Mortality, Housing, Nutrition, Water Supplies.

The *infant mortality* of Batavia has been carefully studied. In the period 1934–36 there were 12,000 native infant deaths, representing 300 per thousand of the live births, but in the poorest native class the rate may be almost 500. In the Chinese the rate was 150, in the Europeans 60 in the same period; for comparison, the Amsterdam rate of 30 is quoted. The native rate is one of the highest reported from tropical cities.

The first three months of life are the most critical; the causes of death are many, but malaria is regarded as one of the most important. No doubt, as in other primitive communities, infant diarrhoea and congenital syphilis are also important.

Houses are made usually of wood, bamboo and palm leaves, but brick is used for the larger dwellings. Doors and windows are insufficient to provide adequate light and ventilation. Overcrowding is not common in rural areas, but occurs in towns.

House improvement is therefore desirable, but is only considered urgent when there is direct danger of disease, as in the case of plague. The Government supports schemes for good housing by giving financial help and by expropriating land for building.

The *nutrition* of the natives is not good, their diet consists largely of carbohydrate foods, is poor in fats, and contains little animal protein though there is a considerable amount of vegetable protein. Rice is the staple food and is largely cultivated. The peasants use a crude form of husking in which the pericarp and embryo (*i.e.* those parts of the grain which contain vitamin B_1) are not removed. This rice is therefore satisfactory, but in other communities the practice of eating polished rice is common.

Maize is used in some areas, but in parts of Java it is the custom to remove the pericarp and give it to the poultry, and to wash the endosperm in running water for three days, a process which removes the vitamin B_1. In Madura, however, the pericarp is eaten as porridge, with good results. Other starchy foods are sweet potatoes (which contain vitamin A), cassava, sago (in Amboina and the Moluccas) and cane sugar. The Government encourages the cultivation and use of soya bean. Peas and beans (gram) are extensively eaten and provide protein (but the djenkol bean sometimes causes damage to the urinary system, see this *Bulletin*, 1936, v. 33, 724; 1942, v. 39, 490); vegetables of several kinds are grown, and are commonly taken as soup.

A considerable amount of fish is eaten in coastal districts, but milk, meat and eggs are little used except by the wealthy or at feasts. Some animal protein, however, is eaten in the form of bee larvae, grasshoppers, frogs and snails, which are appreciated by some of the natives. Coconut oil and ground nuts supply fats, and the latter contain vitamin B_1. Chillies, rich in vitamin C, are used.

Two native foods may give rise to fatal poisoning; they are prepared by allowing moulds to act on ground nut cake (Ontjom) and coconut cake (Bongkrek).

Fruits are usually eaten raw and include the egg plant, the Goa bean and the mango.

The use of tea and coffee is general.

The diet of the native population is deficient in several respects; sometimes it is deficient in quantity, and hunger oedema is not unknown, though it is rare. But the more widespread deficiencies are not of bulk or quantity of total food, but of the vitamins; deficiency in vitamin A is perhaps the most common.

Deficiency of vitamin B_1 is reflected in the disease *beriberi*, which, though it does not now occur in the epidemic form it once assumed, is still seen, and, paradoxically, is often found in the wealthy. It is a disease particularly associated with the eating of polished rice or of sago, and only affects those taking a diet more than two-thirds of which consists of carbohydrate. It is seen in cities, where imported rice is eaten; it is not common in rural districts where the natives only partially clean their rice. It was common during the years of the rubber boom, when agriculture was neglected and rice was imported into Sumatra. In Chinese women it is seen during pregnancy in an acute form, and is due then to the custom of feeding on a diet almost exclusively of polished rice. It tends to break out when maize crops and fishing are poor and when the people are thrown back on to a diet too exclusively composed of rice.

Treatment with preparations of concentrated vitamin B_1 is effective, but the essence of the problem of this disease is prevention, which resolves itself chiefly into the elimination of polished rice, with the substitution of red, or partly polished, rice. The Government encourages the cultivation of leguminous plants and the distribution of red rice and green gram, all rich in vitamin B_1. In controlled populations, for instance in prisoners, the use of polished rice is not permitted. In 1939, beriberi caused 3·41 per cent. of deaths in hospital.

Pellagra is seen sporadically, usually in those who suffer from chronic intestinal disease; in this case the essential fault is, probably, inability to absorb the vitamin which may be present in the food.

There is no evidence of deficiency in vitamin C in the rural population, and scurvy is not seen in them. *Rickets* is not reported. Of all the vitamin deficiencies, that of vitamin A is probably the most common.

Deficiency of iodine in water and food is observed in the mountainous regions of Gayo Loeös, the Diëng plateau, Kediri and Bali. In these districts goitre is common, but the general health of the people is not seriously affected.

In general the natives have excellent teeth.

The water supplies of the rural population consist of rivers, streams, wells and collections of rain water, and are not usually controlled. They are therefore dangerous and are the means of transmitting intestinal diseases. But there are 281 plants for the supply of good and protected water, 138 of which exist in Java. In Sourabaya the controlled supply was instituted in 1903; in Batavia in 1922. In both the result was a decrease in the incidence of typhoid fever.

The provision of safe water is one of the chief measures necessary to good health. In the Dutch East Indies, rivers and streams, in addition to providing drinking water, receive the refuse of man and animals; the danger is evident. Wells are usually shallow and receive not only contamination introduced directly from the hands or feet of the users, or from utensils employed, but contaminating material deposited on the ground nearby, which may either be washed directly into the water, or may reach it by percolation through the soil.

The *disposal of sewage* on a large scale is possible only in the larger towns. Elsewhere, cesspits, septic tanks or pit latrines are used, but in the rural districts the natives commonly defaecate into streams or indiscriminately on the soil. It has already been pointed out that water for drinking is taken from streams and that wells may be contaminated with material from the surface of the ground in the vicinity; they may also be contaminated by percolation from cesspits. The diseases caused by such contamination will be dealt with below.

In some places a solution of this problem is sought in methods whereby excreta are collected and, by a system of composting, are converted into a manure which may be used to enrich the soil. When properly carried out, this method is harmless, and during the process of composting, which consists of packing the excreta and maintaining it in a moist condition, together with vegetable refuse, the organisms which are associated with human disease are destroyed by the heat spontaneously generated in the mass. After a few weeks the material is converted into an odourless compost of high agricultural value.

This process may be exceedingly useful, but for success demands careful attention to the details of routine in periodical turning of the mass, and therefore the human factor is involved. In China and neighbouring countries human faeces are used as a fertilizer without any treatment, and have acted as a potent agent in the spread of disease. Efficient composting is safe from this danger, but it is not difficult to imagine that a native who is, on the one hand, warned of the danger of spread of disease through human faeces, and on the other is taught that the same excreta can, after undergoing a simple process, safely be used in the cultivation of human food, may be somewhat confused.

The *disposal of refuse* is a problem only round the towns, since rural refuse is easily burned. The dustbin system is used in towns and refuse is dumped in the manner adopted in Europe. Stable refuse, however, presents a problem as yet unsolved in many places.

Intestinal Diseases.

Cholera has in the past swept through the Netherlands Indies, as it has through all the neighbouring countries. In 1910 there were 64,733 deaths from this disease, in 1914 just over 1,100, in 1918 almost 10,000, but in 1920 only 17. From 1923 to 1934 there were only 17 known cases.

Cholera is transmitted through the contamination of food or drink with faecal matter, or by direct contact with patients, or through the intervention of flies. It is particularly connected with water supplies, and contaminated water from wells and rivers has in the past played the chief part in the enormous epidemics which have taken place in the East. The protection of water is therefore probably the most important step which can be taken in the prevention of this disease. Though this has been effected in many places in the Netherlands Indies it has not been possible in most rural districts, and to control the disease the public health authorities have in the past resorted to mass inoculation with cholera vaccine, together with strict control of immigrants and pilgrims. It is not easy to determine the efficacy of each of these measures, but the fact remains that in the last 20 years the true classical cholera has not presented a serious problem, though in view of the fact that it is constantly seen in India, Indo-China and China, there is always the possibility of its reintroduction.

In 1938, however, a disease broke out in Celebes which was indistinguishable from cholera but which, on investigation, proved to be due to a vibrio apparently identical with that found in pilgrims at El Tor, on the Red Sea. This El Tor vibrio had never before been found capable of producing disease in man, but there was no doubt of its effect in Celebes, where cases were found up to 1940, though the outbreak was not large.

Typhoid.—In Batavia the cases of typhoid in 1927 were :—Europeans 6·7 per 1,000 of the population, natives 1·1, Chinese 3·8. This high rate in Europeans indicates the danger they run. In 1937 there were about 5,000 reported cases of true typhoid in the Netherlands Indies, and in addition there were 946 cases of paratyphoid A. Inoculation against these diseases should be insisted upon by Europeans.

Bacillary dysentery affects all ages, but is common in children ; this disease and infant diarrhoea are responsible for a high proportion of infant deaths in all tropical countries. Dysentery is constantly present, but increases to epidemic proportions from time to time. About 70 per cent. of the cases are due to the Flexner group of organisms, and the disease is relatively mild, but the severe Shiga type is also found, especially in the Bantam region. In Java and Madura in 1939 there were 5,031 cases of bacillary dysentery with 536 deaths, but these are only the known cases ; many others must go unrecorded. This disease causes about 4·5 per cent. of all hospital deaths.

Amoebic dysentery is much less acute. It is found in many parts, but epidemics rarely occur.

Other Diseases.

Of the diseases caused by worms, *Hookworm infection* is the most widespread in the Netherlands Indies. The common species of hookworm is *Necator americanus*, which is found in 91·8 per cent. of cases, the remainder being due to *Ancylostoma duodenale*.

A consideration of the life-history of the worm makes it clear that spread is a question of bad sanitation. Natives usually deposit their excreta at any convenient spot, and commonly choose shady bushes for the purpose. Plantations offer many opportunities for this practice, and, moreover, the conditions of shade, temperature and moisture are favourable to the larvae. Further, it is in just these places that natives walk barefoot, offering to the larvae the bare skin necessary for their entry into man. On the other hand, faeces deposited in deep pit, or bored-hole, latrines are harmless, though from shallow pits the larvae may be able to reach the ground surface. Latrines whose edges are fouled with faeces, however, are dangerous because bare feet are constantly placed well within range of the larvae, and larvae are attracted by the warmth of human skin.

In the Netherlands Indies hookworm infection is widespread in the rural districts, where 80 to 90 per cent. of the natives may be affected, but fortunately the average number of worms is not high. This means that although minor disabilities no doubt exist, serious illness is not common. This may be a result of the measures of control which have been taken, and which consist of campaigns to encourage the construction and use of proper latrines, and of the fact that most hospital patients, in addition to being treated for their primary illness, are treated by one of the drugs efficient in the elimination of hookworms. It has

been observed that there is a marked decrease in the incidence of this infection where intensive sanitary work has been carried out.

The combination of hookworm infection and malaria gives rise to a condition of severe anaemia, frequently found in some districts.

Other intestinal worms are common, but are less important. Tapeworm infection due to *Taenia saginata* is found especially in Bali. In other places *Taenia solium* is present, and both worms are widespread. Infection with these is rarely serious. *Trichinosis* is also found.

Infection with *Echinostoma ilocanum* is found in Java among the insane who live in colonies, and in Celebes among the people who live round Lake Lindoe. This worm passes one stage of its life in certain fresh water snails, and the second stage either in snails of other species or in fresh water mussels (in Celebes). These snails and mussels are eaten and are often insufficiently boiled, so that the larval forms of the worms are not killed, and proceed to their third developmental stage, in the intestine of man, where they reach maturity. The symptoms caused are slight, some diarrhoea and abdominal pain, and the infection usually dies out in the course of several months. Round Lake Lindoe up to 96 per cent. of the population of some villages are infected.

Infections with *Ascaris lumbricoides* and *Trichuris trichiura* are common, especially in children. They indicate bad sanitary habits, but are not often the cause of severe disease.

Filariasis.—There are two common filarial worms, *Wuchereria bancrofti* and *W. malayi*; the embryo of the latter was first recognized by Brug in the Netherlands Indies. *W. bancrofti* is found in Batavia, the island of Kabaena, Celebes, New Guinea and elsewhere. It is carried by the mosquitoes *Culex fatigans*, *Anopheles sundaicus*, *A. subpictus*, *A. aconitus*, *A. barbirostris* in the more western islands, and in New Guinea (where the mosquito fauna is quite different) by *A. punctulatus typicus*, *A. punctulatus moluccensis* and *A. barbirostris bancrofti*. The breeding places of most of these have been indicated in the sections on malaria—control of breeding is not easy.

Wuchereria malayi is found in Borneo, Batavia and elsewhere in Java (especially in the Serajoe delta), Celebes, New Guinea, the centre of Sumatra and other places. It is carried principally by mosquitoes of the genus *Mansonia*. These mosquitoes have breeding habits quite unlike the mosquitoes already mentioned in that the larvae, developing in water, obtain their oxygen by piercing the roots of certain water plants. This is important because the larvae cannot develop if these plants are not present, and removal of the plants is therefore effective in control; unfortunately some of the mosquito species attach themselves to the roots of swamp trees which cannot effectively be removed. The species of *Mansonia* concerned are *M. annulifera*, *M. uniformis*, *M. indiana*, and they breed largely in association with water plants of the genera *Ipomoea* and (in some places) *Pistia*. In Martapoera (Borneo) infection was found in 22 per cent. of the specimens of *M. annulifera* and in 6 per cent. of *M. uniformis* examined. It is stated that *Anopheles barbirostris* is a vector of *W. malayi* in Celebes and *Anopheles hyrcanus* in Java.

The percentage of people in whom embryos are found varies from 9·3 in part of New Guinea to 42 elsewhere in that island, from 20 in Kabaena to 24·7 in Celebes, from 11·8 in children in Borneo to 32·2 in adult males in the same area. In a minority of cases, however, filarial infection produces elephantiasis and other physical disabilities.

Leprosy.—It is estimated that there are about 50,000 lepers in the islands and that in some areas the incidence reaches 1–1½ per cent. of the population. It is generally accepted that leprosy is a disease transmitted from man to man by direct contact, but that, in general, contact must be prolonged and intimate. For this reason it is a disease commonly transmitted within families.

For the treatment and isolation of lepers there were, in 1939, 49 institutions in the Netherlands Indies, with accommodation for 5,300 patients. The majority of lepers are therefore living at large, and to reduce the risk of infection of healthy persons, the Government encourages the isolation of patients in separate huts in their own districts. Such isolation is usually only partial, but in a disease in which long intimate contact is a feature of infection, such partial isolation may have some value.

Tropical ulcers of the legs are general. They arise commonly from trivial injuries and attain their greatest extent in the malnourished. These ulcers may be enormous, destroying the tissues down to the bone, and even necessitating amputation. They are often found in plantation labourers who are especially prone to the injuries which initiate the process. Much working time is lost through these ulcers, and often needlessly so; if slight abrasions were at once treated, many ulcers would be prevented, but when once they become large, which they do rapidly, cure is a lengthy process.

Yaws.—In the Netherlands Indies surveys of the incidence of yaws have been made; in one of these 20·7 per cent. of the population examined were found to be infected, 8·9 per cent. being in the infective stage, but in some villages the rate was as high as 60·3 per cent. The total number of cases in the islands is therefore enormous.

Treatment with the salvarsan preparations is very effective, especially in the early stages, and the Dutch conduct special and widespread treatment campaigns. In 1927, 731,000 persons were treated, and the Government have adopted the policy of demanding a small payment from those who can afford it, on the ground that this makes the people appreciate more fully the value of treatment.

Venereal diseases are widespread. It is estimated that 10–15 per cent. of the population of Java and Madura, between the ages of 18 and 50, are infected with one or other of these diseases. Syphilis has been found in 4·1 per cent. of Indo-European women, in 7·7 per cent. of Chinese, and in 4 to 6 per cent. of native women in Batavia. In general, gonorrhoea is found twice as frequently as syphilis. Brothels flourish in the cities, and these diseases are much more common in urban than in rural communities. In Batavia 4 to 5 per cent. of the children are syphilitic.

Treatment for syphilis is generally available in hospitals, but three-quarters of the natives who receive treatment do not complete the full course. Further, more than half of the infected people do not attempt to undergo correct treatment, but rely on the many native quack remedies. It is therefore evident that, as in most other tropical countries, the syphilis position is unsatisfactory.

The other venereal diseases, soft sore, lymphogranuloma inguinale and granuloma venereum are seen, but are not nearly so common as syphilis and gonorrhoea. In 1937, 1,281 cases of granuloma venereum were reported; for this, treatment with tartar emetic injections is often effective.

Tuberculosis is prevalent, especially in towns. The common form is pulmonary.

The extent to which infection has spread is indicated by the results of the tuberculin test in Batavia and rural Java where the percentage of positive results is 10 in infants, 35 at ages 5–14, and 68 to 70 in adult life. The number of cases in which *infection* goes on to *disease* is, of course, very much smaller.

That tuberculous meningitis is not rare is shown by the fact that 180 cases were seen in a Batavia hospital from 1932 to 1940. This number constitutes half of the total cases of meningitis admitted. These can only be a fraction of the cases actually existing in the populations.

During 1939 there were 8,677 patients admitted to hospital in the Netherlands Indies for tuberculosis of the lungs, and 1,273 for other forms of the disease. The actual number of cases in the islands must be much higher than these figures indicate. Tuberculosis of the lungs accounted for 10·53 per cent. of all hospital deaths, and other forms for 1·02 per cent. In a survey of 330,000 estate labourers in Sumatra in 1930 it was found that tuberculosis caused more disability and deaths than any other disease except acute respiratory diseases (for instance pneumonia).

Tuberculosis is therefore a serious problem and probably one of increasing importance, and no doubt it is true that the natives do not possess the same standard of resistance as the white races. Other factors influence the extent and spread of the disease, especially the habit of overcrowding in the homes, the poor state of nutrition of many natives, and possibly the presence of hookworm disease and malaria.

To deal with this situation the Dutch have taken certain steps. A tuberculosis study committee has been set up to investigate the position. Instruction in measures of prevention is given in association with the other health campaigns which are conducted, for instance the hookworm campaign. There is continuous effort to improve diet and housing conditions. For treatment and isolation of the patients sanatoria have been built, 4 by Government and 3 by other bodies, while 10 of simpler type are projected. There are 15 consultation bureaux, equipped with X-ray apparatus. The sanatoria are so much in demand that they cannot accommodate all the patients seeking admission, and a system has been developed under which the owners of houses round the sanatoria take in the patients who receive their treatment at the institution.

Smallpox is not now a serious menace. In 1913 there were 35,000 cases with 8,000 deaths, but this high incidence has fallen; in 1927 there were only 400 cases, and since then there have been very few. A vaccine institute was established in 1891 and vaccination is now carried out widely and consistently.

Pneumonia is one of the principal causes of death in both children and adults. It is three or four times as common in the natives as in the Europeans, and the case mortality rate (30 per cent.) is twice as high. In the army it is more important than typhoid, and in prisons in Java it occurs in epidemic form. It is thought that the chilling effect of clothing wet with rain has something to do with the frequency of this disease, and that other diseases, especially malaria, predispose to it. Preventive measures in the general population cannot easily be taken, but from experience gained in South Africa it would seem that the

avoidance of overcrowding in the sleeping quarters in prisons and barracks may lead to diminution of incidence in those communities.

Diphtheria is important. In 1939 there were 786 cases with 106 deaths in Java and Madura. Cases are also seen in the outer provinces. *Scarlet fever* is stated to occur sporadically, and *measles* and *chickenpox* are seen. *Influenza* is seen every year but occasionally assumes epidemic proportions. There are a few cases of *cerebrospinal meningitis* each year, and several cases of *infantile paralysis* have been reported. *Rheumatic fever* has been recorded.

Rabies is reported, and in 1932, 522 persons were treated through the organization of the Pasteur Institute. This does not mean, of course, that all these persons were actually infected. Cases of *rat bite fever* are found from time to time; treatment with the salvarsan drugs is effective. More important than this is *leptospirosis*, primarily a disease of rats and, to a smaller extent, of dogs and cats; it may affect man. It is caused by *Leptospira icterohaemorrhagiae* or *L. bataviae*, which are present in the urine of diseased rats and which may pass through human skin or mucous membranes. In other countries this disease is known as mud, or slime, fever, and is contracted through contact of human skin with mud or water contaminated by rats. Thus, bathing in contaminated water, or working in rat-infested sewers may lead to infection. In the Netherlands Indies the rats concerned are *Rattus norvegicus*, *R. brevicaudatus* and *R. concolor*.

The disease in man may be severe and even fatal, but on the other hand it may be so mild as to be missed. Diagnosed cases are not rare in Batavia, and examination of the blood of 150 healthy persons revealed the fact that 39 per cent. had at some time been infected.

Rhinoscleroma, a bacterial disease which affects the nose and throat, and which is most common in women aged 20–35, is reported from the Batak lands of Sumatra.

Trachoma is frequently seen, and is responsible for much of the blindness which exists. The incidence varies, and some parts of the islands are free from it. A campaign of treatment is carried out by specially trained nurses working under an eye specialist.

Skin diseases are very common, *impetigo* is seen, especially in children, *tinea imbricata*, a skin infection due to a fungus, and other fungous diseases, are reported quite frequently. Infestation with head and body lice is common.

All forms of *cancer* are found.

Insanity is a condition which has necessitated the institution of four large asylums with 8,000 inmates, five observation asylums with 1,100 inmates, and seven observation stations with 316 inmates. The use of *opium* is widespread, but the drug is, in general, used by moderate smokers, and, except in a few cases of gross addiction, does not present a serious problem. Treatment centres for addiction exist. There is little *alcoholism*.

Stone in the bladder or kidney is not uncommon; it has been thought that this condition may be a result of deficiency of vitamin A in the diet.

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MALARIA.

J. NAT. MALARIA SOC. Tallahassee, Fla. 1944, June, v. 3, No. 2, 147–50. **Report on Malaria Terminology from the Committee on Medical Research.**

The Committee on Medical Research of the National Malaria Society suggests that the Society should draw up and adopt a uniform terminology in malaria. A glossary has already been given in the "Report on Terminology in Malaria" published by the Health Organisation of the League of Nations in its *Bulletin*, Vol. 9, No. 2, 1940. The Committee disagrees with some of the terms in the glossary and thinks that better terms could be used. For example, the parasitic periods should be designated prepatent, patent and subpatent, and the clinical periods should be termed incubation, symptomatic and latent (?); induced malaria should be defined as "malaria experimentally contracted" and be divided into (*a*) blood-induced and

(*b*) sporozoite-induced ; the term malaria should be modified by the specific name of the parasite, *e.g.*, *falciparum* malaria instead of aestivo-autumnal, tropical, subtertian, pernicious malaria, &c.

J. F. Corson.

J. Parasitology, 1944, June, v. 30, No. 3, 206–8. **Second Report of the Committee on Terminology of Strains of Avian Malaria** [Huff, C. G., Chairman, Boyd, G. H. & Manwell, R. D.].

Lowe, J. **Some Common Misconceptions of Malaria.** *Indian Med. Gaz.* 1944, May, v. 79, No. 5, 207–10.

Malaria has been widespread and severe in India. Quinine and synthetic antimalarial drugs have been in short supply. Medical and lay contributors to the lay and medical press have made very questionable statements regarding malaria and its treatment. Claims made for specific remedies in advertisements in the lay press and medical journals have been grossly misleading. These are some of the considerations that prompted the writing of this paper, which is intended primarily for those who have relatively little experience of malaria.

The recent high incidence of malaria in its relatively severe clinical forms is explained in part by the movement of bodies of people from one area to another and the carriage of new strains of parasite from one part of the country to another.

The author inveighs against the common idea that untreated cases of malaria in which parasites cannot be found in the blood are quite common ; he has not seen such a case. A thick blood film properly stained and examined remains the most reliable method of detecting malaria infection. A negative result usually rules out an active malaria infection. Sternal puncture rarely, if ever, shows parasites when a good thick film fails to demonstrate them. The presence of parasites does not necessarily mean that they are the cause of the patient's symptoms. A case of meningococcal infection in a patient harbouring malaria parasites is described to illustrate this point. Sound clinical observations will prevent many mistakes.

A protest is made against delaying specific treatment of malaria until the patient has had a few febrile attacks. Unnecessary delay in instituting treatment is criminal.

With regard to the dosage of quinine the author's practice is to give 22½ grains a day for two days followed by 15 grains for five days in *P. falciparum* infections. *P. vivax* infections are treated with 15 grains a day for seven days. Administration should be oral if possible ; if not it should be intravenous. The intravenous dose should not exceed 10 grains and the injection should take at least 10 minutes. In most Indians 6 or 7 grains are adequate. If repeated, the interval between injections should usually be at least six hours. The chief use of intramuscular injections of quinine is in very young children in whom oral or intravenous administration is difficult or impossible.

Norman White.

Sarmento, A. Aspectos médico-sanitários da região de Camacupa. [**Epidemiology of Camacupa.**] *Africa Médica.* Lisbon. 1944, June, v. 10, No. 6, 107–18, 5 figs. [11 refs.]

Camacupa is an administrative district of the Province of Bié, Angola. It lies in lat. 12°08′S., longitude 17°33′E., at an altitude of

1,476 metres, and is crossed by several streams. Vegetation is of poor growth in spite of the fact that in the time of the rains parts become almost lakes. Rice is the commonest cultivation ; others being millet and cassava. The population totals 96,602, of which the blacks constitute 99·1 per cent., and the whites and half-castes each 0·4 per cent. only. The diet is monotonous and the food deficient in protein (especially animal protein), in fats (both animal and vegetal) and in vitamins.

As for diseases, malaria is most common ; in fact Camacupa is to be regarded as an hyperendemic area. In 1940 there were 1,500 cases, among them 8 of blackwater fever, and, in 1941, 1,823 cases, with 3 of blackwater fever. In the whole of the rest of the Province of Bié there were only 4,476 cases of malaria and 12 of blackwater fever ; that is, 25 per cent. of all the cases of malaria and 8 out of 20 cases of blackwater fever in the province in 1940 occurred in Camacupa. One hundred and ninety were seen in children under one year and 504 under five years. Deaths from malaria were few ; in successive years from 1938 to 1941 they were 7, 15, 3 and 16 only. Splenomegaly was examined for in 100 men, 100 children and 137 women and the percentages were 19, 66 and 29·9 respectively. The parasitic index was nineteen ; *P. falciparum* was commonest, 63·1 per cent., *P. vivax* next, 31·2 per cent., *P. malariae* only 5·2 per cent. Other diseases frequently met with were phagedaenic ulcer, especially of the legs, and goitre, which is often seen of a large size. Two of the latter are illustrated and the glandular enlargement in each is almost half the size of the patient's head. *H. Harold Scott.*

FAUST, E. C. **Malaria Mortality and Morbidity in the United States for the Year 1942.** *J. Nat. Malaria Soc.* Tallahassee, Fla. 1944, June, v. 3, No. 2, 79–83, 1 fig.

The malaria mortality in the United States continues to decline. In 1942 only 841 deaths were ascribed to malaria. In that year only eight States had malaria mortality rates exceeding 1 per 100,000 : these were Arkansas 6·0, South Carolina 5·3, Mississippi 3·8, Alabama 3·3, Louisiana 2·6, Florida 2·5, Georgia 2·4 and Texas 1·42. The average malaria mortality rate for the 14 southern States has declined without intermission from 11·2 in 1935 to 2·02 per 100,000 in 1942. Only four counties had rates exceeding 25 per 100,000 ; there were 29 such counties in 1941.

Malaria morbidity data are very incomplete in the United States, as everywhere else. Only in the State of Mississippi does notification appear to be adequately carried out ; here the ratio of malaria deaths to malaria cases was 1 : 378. For the United States as a whole this ratio was 1 : 70. *Norman White.*

HACKETT, L. W. **Spleen Measurement in Malaria.** *J. Nat. Malaria Soc.* Tallahassee, Fla. 1944, June, v. 3, No. 2, 121–33, 1 fig.

The author uses the results of the spleen and blood examination of nearly 40,000 children aged 2 to 12, carried out in winter surveys over a period of years in seven Italian and seven Albanian towns, to demostrate the importance of the spleen survey in sizing up an endemic situation. Over 95 per cent. of the endemic malaria present was revealed by the spleen survey alone. The spleen survey is of the greatest value in measuring the results of control measures and in comparing the

malaria intensity of different places. It is not advocated that microscopic examination of blood preparations should be neglected; it should be carried out where facilities are available. Spleen and blood surveys present different pictures of the same situation; they should be made on the same individuals.

The importance of the spleen survey makes it most desirable that a standard method of examining spleens and recording their sizes should be adopted. The method advocated is not the method commonly associated with the author's name; it is one that is already widely used. The term "spleen rate" should apply to the group examined, "spleen index" to the whole community. The standard spleen rate should apply to children in the relatively non-immune period of life, not over 14, excluding babies. For recording spleen sizes the abdominal surface to the left of the midline from the costal margin to the symphysis pubis is divided by horizontal lines into four equal spaces, two above and two below the umbilicus. Spleens which are palpable only on deep inspiration are number 1 spleens. Larger spleens are numbered 2 or 3 if above the umbilicus, 4 or 5 if below it. The size of the "average enlarged spleen" is significant. It tends to be higher the later the transmission season. One may find a very high spleen rate with an average enlarged spleen of little more than 1; this would indicate a very short, intense period of infection. The "average spleen" is of less account; it takes count of the O or non-palpable spleen. The spleen rate is necessary for its interpretation.

This simple method of recording and averaging spleen sizes has been criticized as inexact, but the estimation of the degree of splenic enlargement by abdominal palpation is a rough-and-ready procedure. The method has stood the test of time and statisticians in many lands.

Norman White.

Laouilheau, R. Indices splénique, plasmodique, gamétique chez les écoliers de Libreville. [**Spleen, Parasite and Gametocyte Indices among School Children of Libreville.**] *Rev. Sci. Méd. Pharm. et Vét. de l'Afrique Française Libre.* 1943, July, v. 2, No. 3, 209–12.

In Libreville, the capital of Gabon, the author examined 1,069 school children from 5 to 15 years of age, in the month of June, the beginning of the dry season. Malaria parasites were found in the blood of 67 per cent. The spleen index was 40 per cent. Of the children examined 145 were half-caste and 924 native. The parasite index of the native children was higher than that of the half-castes, but their spleen index was much lower. Of the infections found 39·44 per cent. were *P. falciparum*, 34·75 per cent. *P. vivax* and 4·02 per cent. *P. malariae*. The remainder were mixed infections. Gametocytes were found in 133 children, 18·5 per cent.

Microfilariae were found in the blood of 51 children, and a trypanosome in one. No spirochaetes were found. *Norman White.*

Yao, Y. T., Wu, C. C. & Pei, Y. S. **Some Epidemiological Factors of Malaria in Mangshih, Yunnan, with Remarks on the Occurrence of Blackwater Fever.** *Chinese Med. J.* Washington. 1943, July–Sept., v. 61, No. 3, 197–211.

This is a record of the results of a malaria survey made during the second half of 1940. Mangshih is a valley between Lungling and Chefang,

near the south-west terminus of the Yunnan-Burma Highway. It is about 1,120 metres above sea level. It has a population of 25,000, mostly Shan, of whom 3,000 live in the town of Mangshih. The climate is mild ; the rainy season begins in April and ends in September.

The spleen rate of children under 13 years of age was 30·7 in the town and 36·1 in the villages. The corresponding parasite rates were 25·2 and 32·1. *P. falciparum* infections predominate, 85 per cent. of the total. Infection rates are much higher among Chinese than among Shans. There is a marked increase of malaria cases in the last three months of the year. *A. minimus*, the chief vector, was most plentiful from September to November.

Sixteen species of *Anopheles* were identified. Of 18,177 adults 12,239 were *A. minimus*, 2,912 *A. jeyporiensis* var. *candidiensis* and 2,494 *A. hyrcanus* var. *sinensis*. Of 19,572 larvae 6,718 were *A. sinensis*, 5,879 *A. annularis* and 4,282 *A. minimus*. The other species were, in comparison, infrequent. Infections were found in one of 1,695 *A. sinensis* dissected, in 3 of 1,363 *A. candidiensis*, and in 195 of 5,883 *A. minimus*, an infection rate of 3·3 per cent. for this species. *A. minimus* shows a much greater preference for human dwellings than the other species : ditches are its favoured breeding place.

Five cases of blackwater fever are reported. The disease is not uncommon in the south-west of Yunnan ; elsewhere in China it is rare. [See also this *Bulletin*, 1941, v. 38, 501, 502.]

Norman White.

Ogborn, R. S., Raper, A. B. & Wright, R. J. **Studies in Malaria in the East African Command. II. A Reconsideration of the Diagnosis of Malaria from other Fevers in East African Natives.** *East African Med. J.* 1944, Apr., v. 21, No. 4, 101–10.

The tendency to regard all fevers among East African natives as malarial, until the contrary is proved, is unjustifiable. In a large military hospital during 9 months there was no death from uncomplicated malaria, but the case mortality rates of other common fevers were, pneumonia 4·1, typhoid 20·3, meningococcal meningitis 15, and bacillary dysentery 0·74 per cent. Of the 155 deaths attributed to these causes, pneumococcal and meningococcal infections were responsible for 120 ; these infections were often rapidly fatal. During the same 9 months post-mortem examinations of fifty patients brought in dead, or dying soon after admission, excluding deaths from violence, showed that 22 had died from pneumococcal infection and 12 from meningococcal infection. If early treatment should appear necessary to avert immediate danger, sulphapyridine should be the drug of choice rather than quinine or mepacrine.

A clinical and laboratory study was made of 512 East African natives admitted to hospital for fever in whose blood malaria parasites were discovered. Of these 384 proved to be cases of uncomplicated malaria. In 116 cases other diseases were responsible for the fever, the finding of malaria parasites being incidental. In 12 cases active malaria coincided with some other disease. The 116 cases in which the malaria parasites were not responsible for the symptoms included relapsing fever, 5 ; typhoid, 7 ; amoebic dysentery, 5 ; lobar pneumonia, 42 ; acute respiratory disease, 28 ; bacillary dysentery, 16 ; miscellaneous, 13.

In determining the significance of positive malaria blood-slides in East African natives attention should be paid to the state of malaria immunity of the patient, the clinical condition, and the intensity of the malaria infection. A native who had spent his boyhood in a region where malaria is prevalent for more than six months in the year was regarded as immune. Parasites were considered to be "few" if an average of less than one per field was found in a thick film of average density, stained by Field's rapid method and examined under a 1/12 in. objective and a ×5 eyepiece. This corresponds to less than 500 parasites per cmm. of blood.

Among the 396 cases of active malaria, in only 16 were no parasites found on the first examination. The second slide was negative in only three of these (one quartan).

Of 229 immune patients harbouring malaria parasites, 37·5 per cent. were not suffering from clinical malaria. The corresponding percentage of 283 non-immunes was 10·6. Of 69 immune patients with only "few" parasites in the peripheral blood 78 per cent. were not suffering from clinical malaria; the corresponding percentage of 28 non-immunes was 43.

Observations show that the administration of quinine in quantity insufficient to control the fever has a negligible effect in eliminating parasites from the peripheral blood.

The examination of thick blood-films stained by Field's method may reveal "toxic granulation," large blue granules in the cytoplasm of the polymorphs. These changes do not occur in malaria but appear early in the course of acute bacterial infections. *Norman White.*

Clark, H. C. **The Age Level for the Peak of Acquired Immunity to Malaria as reflected by Labor Forces.** *Amer. J. Trop. Med.* 1944, May, v. 24, No. 3, 159–61.

If a child has lived in an endemic malaria area a marked degree of tolerance to malaria is gained about the age of puberty but this is not very stable. The peak of relative immunity appears to be attained at an age 12 to 15 years in an inactive population, but from the point of view of an efficient labour force the age groups from 15 to 30 years require almost as much attention as the younger age groups. Immunity is never absolute; fatigue, exposure, underfeeding, or the introduction of fresh strains of parasite, can break any degree of tolerance. The author reproduces figures from his long-continued observations in the Chagres River villages in the Panama Canal Zone and from observations on a Negro labour force from Haiti engaged on sugar plantations in Cuba. The Chagres River villagers have been subjected to antimalarial drug control for many years. By race and life-long exposure they represent people of high tolerance. The consolidated annual records of blood examinations for ten years show that there is a significant incidence of malaria at all age groups. The peak is in the age groups 5 to 20 years, and there is but little difference in the percentage positive blood findings between the two groups 5 to 10 and 10 to 20. These villagers could nearly always tell whether parasites would be found in their blood or not. Similar figures for the inhabitants of the control towns on the Chagres River lake shore show that there is no evidence of a stable degree of immunity before the 20 to 30 age period. The highly tolerant Negro labour force from Haiti showed a sufficient reaction to malaria, after a lifelong exposure to it, to decrease its labour efficiency.

Norman White.

SHUTE, P. G. **Relapse of Quartan Fever after 12 and 21 Years.** *Lancet.* 1944, July 29, 146.

A woman, now 26, was born in Calcutta and came to England when she was 5 years of age; she has not left England since. From 1936 to 1943 she had repeated attacks of fever and suffered occasionally from boils. She was admitted to hospital in 1943 suffering from generalized pains and occasional night sweats. The examination of three thick blood films revealed one *P. malariae* parasite in one and three ring forms in another. A course of mepacrine, 0·3 gm. a day for seven days, effected a cure. There was no enlargement of the spleen.

Diagnosis may not be easy in patients harbouring very few parasites. The author suggests that such cases may not be infrequent in the British Isles after this war. *Norman White.*

GAMMIE, R. P. **Congenital Malaria in England.** *Lancet.* 1944, Sept. 16, 375–6.

A woman returned to England from India in August 1939. Early in October she had an attack of fever which was repeated in 48 hours and her blood showed parasites "suggestive of a benign tertian infection"; this was treated with mepacrine and she remained well until three days before the onset of labour, which occurred on 1st December, 1939. She developed fever on 2nd December [examination of the blood is not mentioned]. The child weighed 7 lb. and was breast-fed for 14 days only. On 3rd January, 1940, the child had fever and *Plasmodium vivax* was found in its blood; there was severe anaemia and the spleen reached to the iliac crest. Treatment with quinine was successful and on June 10th the child weighed 13 lb., and there was no anaemia and the spleen was just palpable. The author saw the child, now aged 4 years, recently and found it very healthy and there was no history of illhealth.

The possibility of infection of the child by mosquitoes in December 1939 can be excluded and the case is regarded as one of congenital malaria. [For other records see this *Bulletin*, 1925, v. 22, 399 & 806; 1926, v. 23, 539; 1929, v. 26, 11; 1936, v. 33, 264; 1938, v. 35, 252; 1942, v. 39, 518, and 1944, v. 41, 354.] *J. F. Corson.*

CRAWFORD, T. **Technique of Blood Examination for Malaria Parasites.** *Brit. Med. J.* 1944, Sept. 9, 348.

There is nothing new in this article, which describes the methods of preparing and staining blood films with Leishman's and Giemsa's stain.

The author points out that many laboratory workers, and others not previously concerned with malaria diagnosis, now find it necessary to become familiar with making and staining films, and diagnosing, not only the malaria parasite, but also the *species* of parasite. There are, however, several points in the article with which to disagree.

It is advised that Leishman's stain should be kept for 10 days in a dark cupboard, followed by a further 7 days in ordinary light before the stain is mature. [The reviewer finds that Leishman's stain is mature within 24 hours of its being prepared. That the distilled water should have a *p*H of 7·2 is now generally accepted. This has been stressed by WENYON, JAMES and others and was described by JAMES in *Trans. Roy. Soc. Trop. Med. & Hyg.*, 1929, v. 23, 269; see also this *Bulletin*, 1930, v. 27, 663.]

For staining thin films Crawford uses 10–12 drops of Leishman's stain, which is allowed to act for 30 seconds and then adds at least twice as much distilled water. [This is excessive and wasteful when three, or at most four, drops of stain, diluted three times after the stain has acted for 15 seconds, is sufficient. In hot climates where an alcoholic stain such as Leishman is used, thin films can first be fixed and then stained, using two or three drops of Leishman and diluting a few seconds later with three times the amount of distilled water.]

The author claims that the length of time required for staining varies with different batches of stain. [If by this he means different *brands* of stain, this may be so, but if it is intended to imply that the same brand varies, then it is likely that inconsistent results are due to variations of the atmosphere where staining is carried out and not to the stain itself. Leishman's stain gives better results if staining is carried out in a room where there are no Bunsen burners or gas fires alight.]

It is advised that in an emergency, distilled water can be boiled for ten minutes to remove the carbon dioxide. [It should have been mentioned that water should be boiled in flasks made of hard glass ; if it is soft glass is used the water becomes very alkaline, owing to the soda in the glass.]

Thick films are stained with dilute Giemsa. [No mention is made of Field's stain, which has largely superseded Giemsa and which gives excellent results.] *P. G. Shute.*

MANSON-BAHR, P. **War Malaria and its Treatment.** [Correspondence.] *Brit. Med. J.* 1944, Sept. 9, 350–51.

The writer observes that in the circumstances of the present war, as in the last one, medical officers with little knowledge of malaria are called upon to treat the disease. He mentions some practical points of difference between benign tertian and subtertian infections and discusses their treatment. Pamaquin (plasmoquine) should not be given in the average subtertian case. Intramuscular injections of quinine are practically painless if neutral isotonic solutions, as in " Solvochin " [quinine+phenyldimethylpyrazolone] are used. Cerebral malaria and blackwater fever do not occur in benign tertian and quartan infections, but only when there has been infection with the subtertian parasite. He has found 20 grains of quinine dihydrochloride, given intravenously, sufficient to restore full consciousness in cerebral malaria, but stresses the importance of the rate (2–4 minutes per grain) at which it should be given. It is surprising how little cerebral disturbance remains after recovery from cerebral malaria. Benign tertian malaria seldom presents alarming symptoms, or causes death, but the parasites persist in the body and cause relapses. [A misprint should be noted ; the dose of atebrin musonate injected was, of course, 0·3 gm., not 0·3 grain as printed.] *J. F. Corson.*

KEOGH, Patricia P. & SHAW, F. H. **The Mode of Action of Quinine Alkaloids and other Antimalarials.** *Australian J. Exper. Biol. & Med. Sci.* 1944, June, v. 22, Pt. 2, 139–47, 11 figs. [19 refs.]

The experiments described in this paper were carried out with the object of obtaining information on the mechanism of the action of quinine in malaria [see also this *Bulletin*, 1944, v. 41, 188]. For this

purpose detailed investigations have been made of the pharmacology of certain members of the cinchona group, including quinine, as well as of other antimalarials. [For earlier related work by the authors see this *Bulletin*, 1944, v. 41, 365.] The effect of these substances on smooth and striated muscle, on the circulation and on other systems, was studied. Quinine and certain other substances tested caused a reversal of the action of calcium on muscle, which was believed to be due to the reduction of calcium ions. Various examples are cited, from biological studies, to illustrate the similarity in effects produced by quinine or by lack of calcium ions.

The authors conclude that the antagonism between quinine and calcium is of a physiological rather than of a chemical character. They consider that their experiments support the hypothesis that quinine interferes with growth and reproduction of the malaria parasite by reducing the amount of calcium available in red cells. This hypothesis is discussed in relation to certain known facts of malariology.

[The statement that " malaria is a disease of the young," quoted in support of their calcium theory is true only for communities of endemic areas. It seems to the abstracter that the interesting hypothesis of the authors could readily be tested now that *P. knowlesi*-infected red cells of monkeys can be obtained in large quantities.] *J. D. Fulton.*

FERNANDEZ MARINA, R. **Atabrine, Intravenously and Orally, in Combination with Quinine and Plasmochin in the Treatment of Malaria. Report of 268 Cases.** *Bol. Asoc. Med. de Puerto Rico.* 1944, May, v. 36, No. 5, 220–22.

The author relates his experience in the treatment of malaria among natives and continental skilled labourers and technicians employed in military constructional work in Vieques Island, Porto Rico. Vieques Island is reputed to be one of the most malarious places in the Caribbean. About 65 per cent. of the native population harbour malaria parasites. The treatment adopted was :—

First 3 *days* : Atabrine [mepacrine or atebrin] 0·20 gm. in 20 cc. distilled water, intravenously. Atabrine 0·10 gm. at noon and 6 p.m., orally. Quinine sulphate 0·5 gm. at bed time. (In heavy infections the intravenous atabrine is dissolved in 500 cc. of normal saline).

4*th and* 5*th days* : Atabrine 0·1 gm. orally thrice daily and quinine sulphate 0·5 gm. at bed time.

6*th*–12*th days* : Quinine sulphate 0·5 gm. at bed time. Ferrous sulphate 0·3 gm. after food.

13*th*–16*th days* : Plasmochin [plasmoquine] 0·01 gm. thrice daily after food. For the next 3 or 4 weeks quinine sulphate 0·5 gm. daily at bed time.

The 268 cases so treated included 112 continental patients and 156 natives. There were 129 *P. vivax* and 139 *P. falciparum* infections. There were no deaths. The relapse rate was 3 per cent. for the continental patients and 12 per cent. for the natives. Ten patients had cerebral symptoms. The only symptoms of intolerance to the doses of atebrin used were one case of " ambulatory delirium " in a chronic alcoholic and two cases of urticaria among the natives. Four cases of mild gastritis were ascribed to plasmoquine. *Norman White.*

HAWKING, F. **Histological Effect of Injection of Mepacrine (Atebrin) Dihydrochloride.** *Brit. Med. J.* 1944, Aug. 12, 209–10.

In a previous investigation [this *Bulletin*, 1943, v. 40, p. 823] the author described the histological effects produced by the injection of therapeutic doses of mepacrine methanesulphonate (atebrin musonate) intramuscularly and subcutaneously into rabbits and rats, and compared them with those caused by quinine monohydrochloride. In order to find out if the effects produced by mepacrine dihydrochloride differed significantly from those of mepacrine methanesulphonate, the experiments have been repeated with both salts of mepacrine, and also with quinine dihydrochloride. Account has been taken of the solubility and *p*H value of each solution.

Macroscopic lesions were caused by all three substances in the long-axis of muscles. In the loose muscles of the thigh, there was some swelling of the connective tissue, often with haemorrhagic patches in the adjacent muscles, and a resulting contracture in one case. Subcutaneous injection caused great thickening of the skin, with gelatinous oedema of the subcutaneous tissues, followed by necrosis and scabbing. Microscopic examination of the fixed tissues at intervals following intramuscular injection revealed, in addition, coagulative necrosis and disintegrated leucocytes to be present at the site. Granulation tissue was later formed around the affected area, and contained shrunken muscle fibres, giant cells and new collagen. The swollen connective tissue was infiltrated with polymorphs and mononuclear cells; haemorrhages and albuminous exudate accompanied the necrosis. The superficial layers of adjacent muscles were also affected, and calcification of some muscle fibres occurred later. Subcutaneous injection caused necrosis down to the muscles of the abdominal wall, accompanied by exudation and white-cell infiltration.

The author concludes that the local toxicity effects of the two salts of mepacrine are indistinguishable, and are identical in type with those due to quinine dihydrochloride, but probably less extensive. Mepacrine dihydrochloride is suitable for intramuscular injection, although less soluble than the methanesulphate; since, however, the local damage caused by intramuscular injections of mepacrine is "far from negligible" this route should not be used if it is practicable to give the drug by mouth. Mepacrine should not be given subcutaneously or intravenously. *J. D. Fulton.*

SANTOS, A. B. Profilaxia quimica da malária no serviço geográfico e histórico do exército. Destacamento especial do Nordeste. [**Chemical Prophylaxis of Malaria in the Geographical and Historical Service in the Northeast of Brazil.**] *Rev. Med. Milit.* Rio de Janeiro. 1943, Apr.–June, v. 32, No. 2, 231–44.

This is a record of the results obtained by the administration of iodo-mercurate of manganese and spleen extract, "Antimalarico Lorenzini," as a prophylactic of malaria and in the treatment of chronic cases. The treatment was given to 94 healthy individuals and to 25 sufferers from chronic malaria. The author makes high claims for the value of this preparation, his results confirming those previously published from the north-east of Brazil [this *Bulletin*, 1943, v. 40, 824].

Norman White.

Roy, B. C. & Chatterjee, K. D. **Malaria. A Preliminary Report on the Studies on the Action of 'ANB-61' (a Preparation of Dita-Bark and Quinine) on Cases of Human Malaria.** *J. Indian Med. Ass.* 1944, Apr., v. 13, No. 7, 193–6, 1 chart.

Hollis, M. D. **Modern Malaria Control.** *Amer. J. Pub. Health.* 1944, May, v. 34, No. 5, 494–8.

Corrêa, R. R. Os vetores de malária no estado de São Paulo. [**Malaria Vectors in the State of San Paulo.**] *Arquivos de Hig. e Saúde Pública.* São Paulo. 1943, Sept., v. 8, No. 19, 121–32, 20 figs. on 3 pls. [26 refs.] English summary.

The author describes the six species of anopheles that are responsible for malaria transmission in the State of San Paulo, *A. darlingi*, *A. tarsimaculatus*, *A. strodei*, *A. albitarsis*, *A. noroestensis*, and *A. cruzi*.

A. darlingi has a marked preference for human blood and will on occasion attack man in daytime. Human dwellings are its favoured resting place. It is very readily infected experimentally and during epidemics infection rates of from 4 to 8 per cent. have been observed. The large number of adults that have been captured in dwellings during epidemics give the impression that its density is high, an assumption that is not supported by a search for larvae. It is the chief vector of malaria in the plateau region where malaria is characterized by epidemic manifestations in different places at different times, phenomena which are ascribed by the author to the "nomadism" of *A. darlingi*.

A. tarsimaculatus is responsible for the endemic malaria of the coastal areas of the State. Though commonly found in greater numbers in stables than in human dwellings it feeds readily on man. Experimentally it is readily infected with either *P. falciparum* or *P. vivax*. It has been found naturally infected. It attains a much greater density near the coast than *A. darlingi* ever does in the plateau region. It has a multiplicity of breeding places; it can breed in small casual collections of water in hoof marks, cart ruts and the like.

A. cruzi is a vector of malaria in forest areas, especially where the trees are parasitized by bromelias, which form the chief breeding places of this species.

Further investigations are necessary to determine the importance of *A. strodei*, *A. albitarsis* and *A. noroestensis* as vectors. They are certainly not so important as the two chief vectors *A. tarsimaculatus* and *A. darlingi*. The absence of these two species explains the immunity to malaria which the Valley of the Rio Paraíba do Sul enjoys.

The three plates illustrate the characteristics of egg, larva and adult of *A. darlingi*, *A. tarsimaculatus* and *A. cruzi* respectively.

Norman White.

Cunningham-van Someren, G. R. **Napier Grass, *Pennisetum purpureum*, for consolidating River and Drain Banks in Anti-malarial Works.** *East African Med. J.* 1944, Feb., v. 21, No. 2, 48–53.

For the protection of the banks of drainage canals and other water courses from erosion by flood many types of grass have been used. The creeping and matted types of grass have the disadvantage that they may grow well into the water and form mosquito breeding places.

The author has made extensive trials with the reed grass *Pennisetum purpureum*, Napier, or elephant grass, and reports favourable results. For success the banks should be well sloped and the first row of grass should be at the base of the bank ; other rows should be staggered at one foot spacing. The grass grows rapidly ; after a year or so it will have stems up to 6 feet in length. It is easily propagated by cutting and will grow in most types of soil in Kenya at altitudes up to 7,000 feet. If the stems are allowed to grow, the shade provided may make conditions unfavourable for *A. gambiae* breeding. The grass has an economic value ; it provides fodder for cattle and the stems can be used for fences and hurdles. *Norman White.*

MURRAY, W. C. & KNUTSON, H. **Airplane Dusting with Paris Green for Control of** *Anopheles quadrimaculatus* **Say in Water-Chestnut covered Areas of the Potomac River during 1943.** *Pub. Health Rep.* Wash. 1944, May 5, v. 59, No. 18, 573–83, 6 figs. (4 on 2 pls. & 1 map).

Water chestnut, *Trapa natans*, first observed in the Potomac River in 1919, spread rapidly ; in 1940 it partially or completely covered every bay and cove along the Potomac from Washington, D.C., to Quantico, Va. Its mat-like growth hinders navigation and affords ideal breeding conditions for *A. quadrimaculatus*. Six military establishments are adjacent to water-chestnut-covered river areas. During 1943 these areas were regularly treated by airplane dusting, once a week from July 4th to September 19th. The total area dusted varied from 3,764 acres during the first week to 1,140 during the last week of this period. The insecticidal dust used was Paris green 1 part mixed with powdered soapstone 4 parts by volume. The average application per acre was 5·4 pounds of the mixture for each treatment. The total cost was $1.20 per acre per application.

The systematic capture of larval and adult *A. quadrimaculatus* in treated and untreated control areas showed that the dusting was highly successful. The control of *A. quadrimaculatus* effected varied from 89·8 per cent. at Fort Washington to 99·7 per cent. at Stump Neck, with a mean of 96·9 per cent. for all six military establishments.

Norman White.

DE ANDRADE, G. C. O Verde-París na campanha contra o *Anopheles gambiae* no nordeste brasileiro. [**Paris Green in the Campaign against** *Anopheles gambiae* **in North-east Brazil.**] *Bol. Oficina Sanitaria Panamericana.* 1944, Mar., v. 23, No. 3, 210–23. [18 refs.]

This is a very comprehensive account of the mannner in which Paris green was used as a larvicide in the eradication of *A. gambiae* from north-eastern Brazil. To it is credited the success of the campaign. In all 261,294 kilogrammes of Paris green were expended. When used dry it was applied in a concentration of one per cent. ; this was the method preferred. When a liquid or damp diluent was used the concentration was 0·45 per cent. The size of the Paris green particles is important. The type " Airfloat," with particles averaging 2 microns in diameter, gave the best results ; it was lethal for from 82 to 100 per cent. first stage larvae. The larvicide mixture was made up near the scene of operations with material obtained locally. All collections of water

which appeared favourable for *A. gambiae* breeding were treated. There was no case of poisoning among animals or man from water treated with Paris green. *Norman White.*

BLACKWATER FEVER.

KEILIN, Joan. **Reaction of Human Serum Albumin with Haematin and Haem.** *Nature.* 1944, July 22, 120–21, 3 figs. [10 refs.]

HEILMEYER, *Deut. Arch. Klin. Med.*, 1932, v. 173, 128, and FAIRLEY and BROMFIELD [this *Bulletin*, 1934, v. 31, 176; 1937, v. 34, 841; *Trans. Roy. Soc. Trop. Med. & Hyg.*, 1938, v. 31, 374] discovered independently that haematin forms with the albumin of human or simian plasma a protein-haematin compound with a characteristic spectrum differing from both methaemoglobin and parahaematin. This pigment, unlike methaemoglobin, does not react with H_2S, NaF, H_2O_2 or sodium azide. It was named methaemalbumin by Fairley, and the product formed from it by $Na_2S_2O_4$ reduction was called haemalbumin.

In the present work, it is pointed out that haemalbumin resembles most closely caffeine-haem, in which caffeine is linked not to the iron but to the porphyrin alone of the haem molecules. For this reason the designation " haematin-albumin " is preferred to " methaemalbumin " for the pigment formed by mixing alkaline haematin and serum albumin or plasma. In pigments of the haemoglobin class the protein is linked to both porphyrin and iron.

On passing CO through a solution of haem-albumin, its spectrum changes to that of CO-haemochromogen, in which protein is linked to iron. This is explained upon the grounds that, as in CO-haem, the addition of CO increases the affinity of the iron atom for nitrogenous substances. Whether the protein component of the CO-haemochromogen is native or denatured albumin cannot be decided, since small quantities of denatured protein appear to be present in all preparations as evidenced by the first appearance of a faint haemochromogen spectrum when human plasma or serum is added to an alkaline solution of haem.

The author summarizes the types of compound which proteins may form with haem (Fe++) as follows :—

1. Haemochromogens. Usually formed by denatured protein linked to the iron.
2. Haem-albumin consisting of native albumin linked only to the porphyrin of haem and analogous to caffeine-haem.
3. Haemoglobins in which the protein is combined with both the porphyrin and iron of haem. *C. Rimington.*

TRYPANOSOMIASIS.

BOMFORD, R. R. **Trypanosomiasis in a European treated with Pentamidine.** [Memoranda.] *Brit. Med. J.* 1944, Aug. 26, 276–7.

The patient, a European aged 32, had lived in a tsetse-fly infested area of Sierra Leone from April 1942 to May 1943 and, apart from malaria,

had remained well. In May 1943 his cervical, axillary and inguinal glands became swollen and painful for a short time. In the following month he returned to the United Kingdom and then had fever, occasional headaches and swelling of the face in the mornings. In the middle of September he reported sick, thinking he had malaria, and an examination of his blood showed scanty rings of *Plasmodium falciparum* and a few trypanosomes. A blood count showed: erythrocytes 3·7 millions per cmm.; haemoglobin 78 per cent.; leucocytes 12,500 per cmm.—neutrophiles 70 per cent., lymphocytes 21 per cent., monocytes 4 per cent. and eosinophiles 5 per cent. Puncture of a cervical gland showed a few trypanosomes. He had irregular fever, to 100·5°F., pulse rate 100–110, slight enlargement of superficial lymph glands, and puffy swelling of the lower eyelids.

Treatment.—Pentamidine isothionate was given intravenously as follows: 100 mgm. in 5 cc. of water on two successive days, then 150 mgm. daily for eight days; an interval of five days; then 200 mgm. followed by nine injections of 300 mgm. each, over a period of 14 days—making a total of 4·3 gm. in 39 days. The injections caused distressing palpitation, a sense of pressure in the head, and itching of the skin, and were therefore given very slowly; there was no fall of blood pressure and the urine remained normal throughout.

Result.—Improvement was rapid and when last seen [date not given] the patient felt quite well. The erythrocyte sedimentation rate improved from 81 mm. in one hour, before treatment, to 22 mm. at the end of treatment, and 5 mm. three months later; the cell count and haemoglobin content became normal. The cerebrospinal fluid was examined for total protein content, cell content, and by the Nonne and Pandy tests for globulin, the Lange gold curve test, and the Takata-Ara test; before treatment the protein content was 0·035 per cent. and the cells 10 per cmm., at the end of treatment these figures were 0·045 and 22, and 3 months later they were 0·03 and 4 respectively . . . "the patient was allowed to return to duty without further treatment and was advised to have his C.S.F. examined in six months' time." [No inoculation of animals seems to have been made but the trypanosome was probably *T. gambiense*. Further reports on the cerebrospinal fluid, particularly the protein content, would be of great interest and value, especially if there has been no risk of reinfection. See also this *Bulletin*, 1942, v. 39, 532; 1943, v. 40, 224, 370 and 589; 1944, v. 41, 466, 928, 929.] *J. F. Corson.*

HARDING, R. D. **Trypanosomiasis treated with Pentamidine.** [Correspondence.] *Brit. Med. J.* 1944, Sept. 30, 447.

The article by BOMFORD [above] reporting the treatment of a case of sleeping sickness by pentamidine has prompted the author to discuss the very important question of the choice of the most suitable drugs for the treatment of this disease. He has had considerable experience of Gambian sleeping sickness in Nigeria and Sierra Leone and has records of 2,000 cases, of which 192 were treated with pentamidine. He considers that the infection in Bomford's patient had passed beyond the first stage. In the 192 cases referred to the dosage of pentamidine varied from 8 to 12 daily doses of 50 to 100 mgm.; no appreciab difference in curative effect was noted between the higher and th lower dosages.

In early cases with a normal cell-count of the cerebrospinal fluid [presumably normal protein content also] pentamidine was about as effective as antrypol [Germanin] followed by tryparsamide, but was less toxic "than some of the antrypol-tryparsamide combinations." In late cases, however, pentamidine was much inferior; where the c.s.f. contained over 100 cells per cmm. before treatment, about 45 per cent. of the patients died within a year, and, of the survivors, about 75 per cent. had definitely abnormal cerebrospinal fluid when examined more than a year after treatment. The corresponding figures for antrypol plus tryparsamide were about 13 per cent. of deaths and 20 per cent. of abnormal c.s.f. It is the general experience in Nigeria, the Gold Coast and the Belgian Congo that pentamidine is of very little value once the central nervous system has been infected; in Sierra Leone the dividing line lay around 10 to 15 cells per cmm. [the author does not give figures of total protein which SICÉ (this *Bulletin*, 1930, v. 27, 820) and FAIRBAIRN (*ibid.*, 1934, v. 31, 559) consider more important than the cell count].

The author regards it as unjustifiable to treat any case of trypanosomiasis with pentamidine alone without first making a lumbar puncture [it is perhaps advisable to sterilize the blood before making a lumbar puncture in case a vein is punctured] and unjustifiable to treat it with pentamidine if the c.s.f. shows more than a very slight degree of abnormality. Excellent results were obtained, however, in 53 cases, some of which were late cases, treated with a combination of 5 doses of 100 mgm. of pentamidine and 5 doses of 2 gm. of tryparsamide, given concurrently; either the two drugs were given together at 5-day intervals, or tryparsamide was given at 5-day intervals and pentamidine daily, the whole course lasting 3 weeks. The author thinks, however, that the well-tried combination of antrypol and tryparsamide is the best for routine treatment of cases with any involvement of the central nervous system.

The 2,000 cases referred to were re-examined more than a year after treatment and the author makes the comment that other cases hitherto reported, which were treated with pentamidine, have mostly not been followed up long enough—at least a year is needed to be sure that a patient is cured.

The dosage used by Bomford, nearly 4 mgm. per kgm., is high and the author doubts whether such high doses are more effective than smaller ones in sleeping sickness. *J. F. Corson.*

VANDERPLANK, F. L. **Studies of the Behaviour of the Tsetse-Fly (*Glossina pallidipes*) in the Field: the Attractiveness of various Baits.** *J. Animal Ecology.* 1944, May, v. 13, No. 1, 39–48, 1 fig.

Various animals—ox, dog, domestic pig, sheep, goat, baboon, porcupine, lion, serval cat, jackal, kudu, reedbuck, and Thomson's gazelle—were led along a path, about 2 miles long, cut in a thicket in which tsetse flies (*Glossina pallidipes*, Aust.) were living, the object being to compare the relative attractiveness of these animals to the tsetse flies. Direct comparisons could not be made in these moving rounds because the leading animal would attract all the flies, so the walks were taken twice a day. Some screens, unscented and scented by bush-pig, were also used for comparison. Direct comparisons were,

however, made by tethering two animals, 50 yards apart in the thicket, interchanging their positions hourly. This method was better than the walks because the flies that were caught on the tethered animals had come to feed and were not merely attracted by a moving object ; other factors that might affect the accuracy of the figures were also eliminated in the stationary method. The results are shown in tables, which give the numbers of tsetse flies caught on the different animals, and the numbers are treated by statistical methods to indicate their " significance " or otherwise.

The author concluded that the animals were attractive to *Glossina pallidipes* in the following order : (1) the dog, ox and porcupine were the most attractive ; (2) domestic pig and screens ; (3) sheep, goat, man and baboon ; (4) serval cat, lion and jackal. The other animals did not provide sufficient data.

The author refers to observations by Swynnerton [this *Bulletin*, 1921, v. 18, 22], Harris [*ibid.*, 1931, v. 28, 524], Lloyd [*ibid.*, 1936, v. 33, 653], Jack (*Bull. Entom. Res.*, 1941, v. 31, 407), and Potts (unpublished). *J. F. Corson.*

Wood, S. F. **An Additional California Locality for *Trypanosoma cruzi* Chagas in the Western Cone-Nosed Bug, *Triatoma protracta* (Uhler).** [Research Notes.] *J. Parasitology.* 1944, June, v. 30, No. 3, 199.

Fifty-seven of a collection of 188 *T. protracta* were found naturally infected with *Trypanosoma cruzi* near Fallbrook, San Diego County, California.

Balcells, L. Observaciones sobre la enfermedad de Chagas en Tacuarembo. Lesiones chagásicas de inoculación seudocarbunclosas. Con anotaciones de R. V. Talice. [**Chagas's Disease in Tacuarembo : Inoculation Lesions simulating Carbuncles.**] *Arch. Uruguayos de Med., Cirurg. y Especialidades.* 1940, Nov., v. 17, No. 5, 481–91. [Bibliography.]

Sullivan, Thelma D. **Viability of *Trypanosoma cruzi* in Citrated Blood stored at Room Temperature.** [Research Notes.] *J. Parasitology.* 1944, June, v. 30, No. 3, 200.

Trypanosoma cruzi, present in citrated blood from infected guineapigs which was stored at room temperature for 257 days, not only remained viable, but actually multiplied. It is considered that examination of citrated blood is satisfactory for the diagnosis of Chagas's disease, even when it has been stored at room temperature. The examinations included culture on Kelser's blood agar slope. *Charles Wilcocks.*

LEISHMANIASIS.

Costa, O. G. Polipo da leishmaniose. [**Nasal Polypus due to Leishmaniasis.**] *Brasil-Medico.* 1944, Mar. 18 & 25, v. 58, Nos. 12 & 13, 88–9, 1 fig.

The patient, an adult from the State of Minas Geraes, showed evidence of old leishmanial ulceration of the legs, and complained of nasal obstruction. A polypus was seen attached to the nasal septum, which, below the attachment, had been widely perforated. The polypus presented in both nostrils ; it was reddish, rounded, soft and its surface

was somewhat irregular. [Nothing is said of how the diagnosis was confirmed.] The author notes that the condition has been described before, but that it is a rare manifestation of American leishmaniasis.

Charles Wilcocks.

PARROT, L. Notes sur les Phlébotomes. XL. Sur *Phlebotomus* (*Prophlebotomus*) *minutus* Rondani et sa variété *parroti* Adler et Theodor. [**Concerning** *P. minutus* **and its variety** *parroti*.] *Arch. Inst. Pasteur d'Algérie.* 1943, Mar., v. 21, No. 1, 38–50, 6 figs. [36 refs.]

PARROT, L. & GOUGIS, R. Notes sur les Phlébotomes. XLI. Sur *Phlebotomus roubaudi* Newstead, 1913. [**Concerning** *P. roubaudi*.] *Arch. Inst. Pasteur d'Algérie.* 1944, Mar., v. 22, No. 1, 40–46, 3 figs. [13 refs.]

PARROT, L. & MARTIN, R. Notes sur les Phlébotomes. XLII. Sur *Phlebotomus larroussei* var. *canaaniticus*. [**Concerning** *P. larroussei* **var.** *canaaniticus*.] *Arch. Inst. Pasteur d'Algérie.* 1944, Mar., v. 22, No. 1, 47–51, 3 figs.

PARROT, L. Notes sur les Phlébotomes. XLIII. A propos de *Phlebotomus mascitii* Grassi. [**Concerning** *P. mascitii*.] *Arch. Inst. Pasteur d'Algérie.* 1944, Mar., v. 22, No. 1, 52–4, 3 figs.

PARROT, L. & MARTIN, R. Notes sur les Phlébotomes. XLIV. Phlébotomes de Djibouti. [*Phlebotomus* **of Jibouti.**] *Arch. Inst. Pasteur d'Algérie.* 1944, Mar., v. 22, No. 1, 55–9, 2 figs.

PARROT, L. & CLASTRIER, J. Notes sur les Phlébotomes. XLV. Présence de *Phlebotomus perfiliewi* en Tunisie. [*P. perfiliewi* **in Tunisia.**] *Arch. Inst. Pasteur d'Algérie.* 1944, Mar., v. 22, No. 1, 60–62. [18 refs.]

FEVERS OF THE TYPHUS GROUP.

CROFTON, J. & DICK, J. C. **Clinical and Pathological Aspects of Typhus in the Middle East Forces.** *J. Roy. Army Med. Corps.* 1944, July, v. 83, No. 1, 1–6.

This paper deals only with the clinical features of 67 cases of typhus fever treated in a large British Military Hospital in the Middle East between March 1942 and October 1943. All but one of the patients were Europeans. In 47 of the cases Rickettsia-agglutination tests carried out by C. E. VAN ROOYEN [see this *Bulletin*, 1943, v. 40, 895] indicated that 28 were murine, probably flea-borne ; and 19 epidemic, probably louse-borne. Of the former group five were severe and one was fatal ; of the latter 15 were severe and one was fatal. In the remaining 20 cases the reaction was not carried out or it gave an indefinite response. The Weil-Felix reaction was positive in all the cases.

The mode of onset was not characteristic ; although nearly all the patients described it as sudden few of them sought admission till the 3rd or 4th day.

The duration of the fever was 4–9 days in five cases ; 10–15 days in 41 ; and 16–24 days in 21, of which 13 were epidemic in type.

Defervescence was by crisis in about half of the epidemic cases and in rather less than one-third of the murine. In all the 9 cases in which the fall by lysis began before the 9th day the disease was murine.

In 70 per cent. of the cases the rash first appeared on the 4th to the 6th day ; it was completely absent in one very mild case. It was often observed on the palms as well as on the trunk and extremities ; sometimes there was a tendency to papulation. Subcuticular mottling was often absent in mild cases ; petechiae appeared in the centre of the macules in severe cases. On the whole the intensity of the rash was in proportion to the severity of the attack.

Tremors, especially of the lips and tongue, occurred in 30 per cent. of the cases, usually about the 7th day ; and in three-fourths of the patients with pronounced tremor the attacks were severe or fatal.

Torpor was a feature of all the severe attacks, its onset was usually between the 7th and 9th day. In eight severe cases there was temporary deafness, beginning on the 7th or a later day.

The spleen was palpable in 30 cases, usually from the 5th day, and often for only one or two days. Râles or crepitations, with cough, were observed in 30 cases, of which 20 were severe. One patient developed pneumonia and another a lung abscess.

Diarrhoea occurred in nine severe cases. There was incontinence of urine, " often also of faeces," in 11 cases, of which four were fatal.

Early in the attack the leucocyte count, though higher on the average than in enteric, was of little diagnostic value.

Great loss of weight, often amounting to 2–3 stones, was a feature of all the severe attacks.

Patients were kept flat in bed and were turned frequently on alternate sides to prevent hypostasis. An intake of fluid of five to seven pints, according to the weather, was enforced, and great importance was attached to a generous diet, with two-hourly feeds of milk and egg flip, supplemented by minced chicken or fish, vegetable purées, eggs, bread and butter, milk puddings, fruit juices, and marmite.

Sulphonamides when given for chest complications seemed to make the patients " more toxic."

Reliable diagnosis was seldom possible before the appearance of the rash, and even then might be difficult apart from the Weil-Felix test. Blood culture for enteric organisms should be done in all cases. Murine cases were milder on the whole than epidemic, but could not be differentiated from them solely on clinical grounds. Of the five fatal cases, three were diagnosed as epidemic, one as murine, and one as of uncertain type.

High-titre Weil-Felix reactions were regarded as diagnostic of typhus ; only one exception to this rule was encountered, a case of paratyphoid B. [The diagnostic titre is referred to as " 14 : 80 " ; presumably 1–480 is intended.]

The post-mortem findings in four cases are described ; varying numbers of microscopical haemorrhages and typhus nodules were found in the brain.

[The fatality rate of about 7 per cent. is stated by the authors to be low, but no reference is made to the great importance of age as a factor in prognosis.

It is to be hoped that the epidemiological aspects of the disease in the Middle East will be studied to find whether the evidence points to flea transmission in the cases in which the Rickettsia-agglutination reaction is of the murine type. In war conditions every case of typhus fever will rightly be handled as one of louse-borne type unless there is proof to the contrary, but if in the cases in which the titre of agglutination to *R. mooseri* is higher than that to *R. prowazeki* the infection

should turn out to be incapable of transmission by lice from man to man, the control measures would be directed against the rat instead of the louse.

The paper should serve the purpose for which it was intended, ***viz.***, to emphasize the variability of the disease and to correct the wrong impression conveyed by some text-book descriptions.]

John W. D. Megaw.

NEUJEAN, G. Enquête sur une épidémie de typhus exanthématique (1939–1940). [**An Enquiry into an Epidemic of Exanthematic Typhus Fever (1939–1940).**] *Rec. Travaux Sci. Méd. Congo Belge.* Léopoldville. 1944, Jan., No. 2, 7–46, 28 charts.

In the Ruanda-Urundi region of the Belgian Congo [in the eastern part of the colony just south of the equator] Weil-Felix tests in suspicious cases were uniformly negative during the years 1936–1938. In the area typhoid fever was endemic and there was widespread infestation with lice and fleas.

Early in 1939 there were two cases in which *Proteus OXK* was agglutinated at a titre of 1–160. Towards the end of the year steadily-increasing numbers of sera from different localities agglutinated *OX19* and an enquiry was started.

Rickettsiae were isolated from lice collected from the patients, and although the organisms were completely non-orchitic they were regarded as murine because of their low virulence to guineapigs.

Rickettsiae were also isolated from the blood of a patient who died on the 6th day ; this strain was similar to the louse strain except that two of the guineapigs used in passages had slight orchitic reactions.

Sixteen pooled suspensions, each of which was made from the brains of 10 different groups of rats caught in the affected area, were used for the intraperitoneal inoculation of 16 guineapigs. Six of the guineapigs reacted ; five of them developed orchitis, but from only two were Rickettsiae recovered, and one of the strains was consistently orchitic.

Between 1.12.39 and 1.5.40 as many as 902 sera of patients gave positive Weil-Felix reactions ; the titres did not exceed 1–320 in 93·6 per cent. of the patients. The titres with *OX19* and *OX2* were the same in all cases, and the reaction with *OXK* was negative in all. In a number of patients the reactions were tested repeatedly and it was found that they became negative within 10 days or so. This observation is difficult to reconcile with the further observation that every one of 28 healthy persons with no history of illness gave positive reactions, and in 22 of these the titre was 1–160 or 1–320. These tests were carried out on persons in a highly-infected area and apparently at the height of the epidemic conditions.

The percentages of positive reactions and the average titres of the agglutination increased progressively during the first two months of the period, and then declined *pari passu* with the decline of the epidemic.

Typhoid fever was prevalent in the area, and among 680 sera subjected to the Widal and Weil-Felix tests 241 reacted to both ; of these, 51 reacted in higher titres to the Widal than to the Weil-Felix test, and 59 reacted at the same titre to both tests. In a number of the cases in which repeated tests were made it was found that the Widal reaction usually became positive later and negative earlier than the Weil-Felix. The Widal reaction was regarded as anamnestic and as significant only in the cases in which the titre was higher than that of the Weil-Felix reaction.

The author concluded that he was dealing with an epidemic of murine typhus fever of low virulence with rapid and widespread diffusion over a wide area. He believed that practically the whole of the population became infected and that most of the attacks were symptomless. The flea was regarded as the vector but the louse was also thought to play a part. There was practically no mortality, and the author felt justified in recommending the cessation of severe measures of control which were being instituted in the area : in fact he believed that the epidemic established a valuable degree of immunity against the risk of louse-borne historic typhus. *John W. D. Megaw.*

JADIN, J. Présence de typhus exanthématique murin à Coquilhatville. [**Murine Exanthematic Typhus Fever in Coquilhatville.**] *Rec. Travaux Sci. Méd. Congo Belge.* Léopoldville. 1944, Jan., No. 2, 47–51.

It is stated that apart from the isolation of murine Rickettsiae described by NEUJEAN in the paper abstracted above, there has been no evidence of the occurrence of murine typhus in the Belgian Congo. In the typhus epidemic of 1934 in Ruanda-Urundi the strain isolated was described as being of the epidemic type.

In February 1940 the author isolated an orchitic strain of Rickettsiae from a pooled suspension of the brains of nine rats (*Mus rattus rattus*) captured in the port and native quarter of Coquilhatville [situated in the interior of the Belgian Congo, on the equator and on the bank of the Congo River]. The strain ceased to be orchitic after 6 passages through guineapigs.

Two other orchitic strains were isolated from rats in the same locality ; one of these ceased to be orchitic after three passages, the other was passaged 60 times through guineapigs and was orchitic in all but three of the passages. It was also passed 15 times through white mice. Grey rats were susceptible but white rats did not react to infection.

Sera of 160 wild rats were tested ; 27 of these agglutinated *Pr. OX19* at a titre of 1–100.

No cases of typhus fever were detected in the area, and no positive Weil-Felix reactions occurred in sera examined during a period of four years, with the exception of those referred to in the following paper by the same author. *John W. D. Megaw.*

JADIN, J. La fièvre rouge congolaise est du typhus exanthématique murin. [**The Red Fever of the Congo is Murine Exanthematic Typhus.**] *Rec. Travaux Sci. Méd. Congo Belge.* Léopoldville. 1944, Jan., No. 2, 52–96, 24 figs. [22 refs.]

The name *Fièvre Rouge Congolaise* was applied by LEFROU in 1927 [this *Bulletin*, 1928, v. 25, 529] to a peculiar kind of fever in the French Congo. The author quotes a definition of the disease, or rather group of diseases, as a fever characterized by a rash and polyadenitis, but he adds that these features are of variable occurrence, so that no clear indication is given of the criteria on which a diagnosis of the fever is based.

The disease is stated to have an almost negligible fatality rate, and among the 20 cases described the rash was absent in 9, and polyadenitis

in 10. It is also stated that some of these cases were not diagnosed as red fever and others were regarded as of a doubtful nature.

[From the clinical details and the fever curves it may be stated that the features of cases are compatible with a diagnosis of flea-borne typhus with a wide range of duration and severity.]

The cases occurred in the Province and town of Coquilhatville which are infested by rats. The Weil-Felix test was performed in 14 cases; in five the reaction was negative; in five, *Pr. OXK* alone was agglutinated, at titres ranging from 1–50 to 1–200; in two there was a low-titre response with *OX19* and *OXK*; in the remaining two, *OX19*, *OXK*, and *OX2* were agglutinated, in one of these the *OXK* titre rose to 1–1,000, and in the other, which was a relatively severe experimental case, the *OX19* titre rose to 1–2,000. Apart from the last-mentioned two cases the titre with any of the *Proteus* organisms did not exceed 1–250.

Extensive laboratory investigations were carried out; some of the chief findings and conclusions were as follows. From all the patients orchitic strains of rickettsiae were isolated by guineapig inoculation. The organisms were highly pathogenic to mice and white rats, and small monkeys were susceptible. Infected material, introduced *per rectum* into lice, caused death in 3–4 days. Lice that had fed on one patient died within seven days, and suspensions made from the guts of these insects caused death in other lice, by intrarectal inoculation, in 3–5 days.

Seven lice, infected per rectum by material derived from the fourth intrarectal passage through lice, were allowed to feed on a volunteer aged 34 years. After 14 days' incubation the volunteer developed a typical attack of typhus fever lasting about 15 days. The *OX19* titre of this patient's serum rose from zero to 1–2,000 and the *OXK* titre to 1–100. Rickettsiae were isolated from the blood by guineapig inoculation, and by intrarectal inoculation of lice. Fifteen lice were fed directly on the patient twice daily for three days; all died within five days and intestinal smears from them were rich in rickettsiae.

[No mention is made of attempts to transmit infection by lice that had been directly infected by biting patients or infected animals, or of any investigation into the infection in rat fleas.]

Reference is made to agglutination tests carried out at Stanleyville and adjacent area of the Belgian Congo, by Dr. Ignace VINCKE who examined 197 sera of patients; 126 of these reacted with *OX19* (56 at titres of 1–800 to 1–12,800); 107 reacted with *OXK* (30 at 1–800 to 1–12,000); and 40 with *OX2* (five at titres of 1–800 to 1–3,200).

An epidemic of typhus fever in Equatorial Africa is mentioned in which other workers found that *OXK* and *OX2* were agglutinated "equally with" *OX19*.

The author concludes that there is a special type of murine typhus in the Congo Basin and that the red fever falls into this category. He states that the transmitting vector is the rat flea, though "as we have shown" the louse may intervene as a vector of infection from man to man.

Cross immunity between the disease and murine typhus has been demonstrated both by the author and by Dr. ORDMAN of the Medical Research Institute at Johannesburg.

The strain has been passaged 60 times through guineapigs without change in its orchitic properties, but after 15 passages through guineapigs it lost its virulence for mice.

[This, and the two preceding papers, are of considerable interest ; they supply additional evidence to show that the typhus fever transmitted by fleas from rats is of frequent occurrence in tropical Africa ; also that neither the orchitic properties of the rickettsiae nor the Weil-Felix reactions can always be relied on to differentiate flea-borne from louse-borne or even from tick-borne typhus. The wide range of variation in the agglutination responses is particularly striking.]

John W. D. Megaw.

TOKAREVICH, K. N. & EPSTEIN, E. F. [**Observations on Typhus in Inoculated Patients.**] *Zhurnal Mikrobiologii, Epidemiologii i Immunibiologii.* Moscow. 1943, No. 12, 18–21, 5 charts. [In Russian.]

In Leningrad in 1942 there were several cases of typhus among the medical personnel, who had been previously inoculated by Weigl's vaccine, and the authors compared the course of the disease in inoculated and uninoculated patients. They had details of 17 cases among them who had been partially or fully inoculated and who were brought into very close contact with typhus patients owing to lack of space, fuel and shortage of staff ; all recovered. The course of the disease was much less severe than usual, especially among the fully inoculated. Headache of varied intensity was the only cerebral symptom ; none became unconscious. The temperature became normal on the 9th or 10th day and in one case on the 7th. The prodromal symptoms in inoculated cases were more accentuated, but developed slower than usual. The rash appeared as usual on the 4th or 5th day, but was more of the roseolar type and rather scanty. In spite of the fact that practically all patients were inoculated more than a year before their illness, the whole course of the disease was lighter and the symptoms were less pronounced than usual. Nevertheless a subcutaneous inoculation of a preparation of the intestines of infected mice (15–30–45 intestines) does not give a full guarantee of immunity against typhus. Typhus may occur even one month after the completion of the course of inoculations. The statement of STERNBERG in his book " Course of infectious diseases in prolonged undernourishment " (1920), that typhus patients, who were admitted to hospitals in an undernourished condition, did not develop a more serious course of the disease than those who were physically fit, has been proved to be correct.

H. W. Swann.

TOKAREVICH, K. N. [**Epidemiological Data on Typhus in Children.**] *Zhurnal Mikrobiologii, Epidemiologii i Immunibiologii.* Moscow. 1943, No. 12, 22–5. [In Russian.]

There were two epidemics of typhus in Leningrad—in 1932 and 1933. Between 1933 and 1941 there were only sporadic cases. This paper deals with the epidemic in the autumn and winter of 1941–42. Leningrad was besieged and blockaded during this time. Owing to the heavy raids and practically continuous bombardment of the city there was a considerable migration of the whole population from one district of the town to the other. During the peak of the epidemic—in January 1942—a great number of children were admitted to children's homes and crèches, where the sanitary conditions were

appalling. That was one of the reasons why there were more cases of typhus amongst children. The following table gives the percentages of the total number of notified cases in Leningrad.

Year	Age in years			
	0–4	5–9	10–14	0–14
	Per cent.	Per cent.	Per cent.	Per cent.
1933	1	4·8	6·9	12·7
1938	0·1	0·7	1·5	2·3
1942	2	5	13	20

This table of the incidence among children shows that school children were more frequently affected than younger ones. Fifty per cent. of all cases occurred amongst children under 14 years of age.

These data show that children have no special resistance to typhus, but there is a general unanimity of opinion that the disease takes a much less severe course in children than in adults. *H. W. Swann.*

Ruiz Castañeda, M. **The Problems of the Protection of Man against Typhus.** *Bol. Oficina Sanitaria Panamericana.* 1944, May, v. 23, No. 5, 411–18. [21 refs.]

The author thinks it reasonable to expect that epidemics of louse-borne typhus may appear whenever conditions favour the transmission of the murine type of Rickettsia by lice.

He quotes three epidemics and two outbreaks in Mexico in which orchitic strains were isolated, and in which the disease was more or less certainly transmitted by lice. On the other hand he states that cross immunity between classical and murine Rickettsiae is incomplete or even negative in experimental animals, so that the conception arose of two distinct types of the organism, differing from each other in their epidemiological features.

In 1930 the author isolated certain strains of the classical type which, after a number of transfers, acquired the characters of the murine strain. Nicolle suggested that these were " intermedial strains " and that their occurrence indicated a stage in the transformation of the murine to the classical strain.

Mooser and his colleagues, in 1934 [this *Bulletin*, 1934, v. 31, 621], transformed a classical strain to a murine one by laboratory procedures, and in 1937 the author and Silva [this *Bulletin*, 1940, v. 37, 259], during an epidemic in Mexico, isolated not only distinct murine and classical strains but also classical strains which became murine-like after transfers through guineapigs. All three strains were recovered from cases in the same hospital, and it was believed that they had a common origin from a single strain, from which they developed by a process of evolution in which the murine and classical strains became " very different from the original strain."

The differences are not regarded as fundamental, but rather as quantitative, as shown by the fact that murine vaccine in large doses protects against classical typhus, though classical vaccine does not protect against murine typhus.

In Mexico a combined vaccine is recommended ; this is prepared, by the method introduced by the author in 1938, from the lungs of

intranasally-infected animals ; the rat is used for the murine element and the mouse for the classical. Cox's method has been found unsuitable in Mexico ; its yield of antigen is relatively small.

Large doses of the combined lung vaccine, injected five or six times at weekly intervals, have given very satisfactory results in protecting laboratory workers engaged in the preparation of lung vaccines ; the reactions are sharp but not so severe as those caused by typhoid vaccine.

The author justifiably objects to the practice of calling the two main strains of organism " epidemic " and " endemic."

John W. D. Megaw.

URRA, I. A. Fleckfieber in Spanien. [**Typhus Fever in Spain.**] *Deut. med. Woch.* 1944, Feb. 4, v. 70, No. 5/6, 60–62, 1 fig.

This report deals chiefly with certain haematological and serological findings in 1,322 cases of typhus fever under the charge of the author in a temporary hospital in Seville.

There was pronounced leucocytosis in 69 per cent. of the cases, the usual count was 10,000–20,000, but in one case it was 32,000. In 11 per cent. there was leucopenia. A definite increase was observed in the myelocytes, especially in severe attacks, and a deviation to the left in the differential count. The lymphocytes were diminished, and eosinophiles were completely absent.

Bone-marrow smears obtained by sternal puncture yielded important information ; the most striking feature was an increase in the myeloblasts, promyelocytes and myelocytes ; there was also a diminution in the red cells and normoblasts.

The Weil-Felix reaction was positive in nearly 100 per cent. of the cases, though sometimes not till the 21st to the 27th day, in the convalescent stage. With an American strain of *Proteus OX2* there was a negative reaction in 38·4 per cent. of the cases that reacted with *OX19*. Positive reactions with *OXK* were very rare, and when they did occur the titre did not exceed 1–10.

Praise is given to a rapid bedside-agglutination test in which a drop of suspension of *Proteus OX19*, stained with Victoria blue, is mixed with a drop of finger blood on a slide. Details are given of the technique adopted by the author's colleagues, but this differs in no essential respect from the test as employed by CASTAÑEDA *et al.* [this *Bulletin*, 1941, v. 38, 448] and modified by BRUMPT.

A treatment for which dramatic results have been claimed by DANIÉLOPOLU was tried and found effective. This consisted in intravenous injections of 250–500 cc. of a solution containing 0·5 gramme of chlorine and 6·5 grammes sodium chloride in 1,000 cc. water. The injections were given once or twice daily in very severe cases in persons over 40, and a reduction of the fatality rate from nearly 20 per cent. to under 9 per cent. was obtained. The results were not so startling as those claimed by Daniélopolu [this *Bulletin*, 1921, v. 18, 305] in the first world war, but the author was strongly impressed by them. No unfavourable reactions were observed, and although the duration of the fever was not affected there was a striking improvement in the neurological manifestations. [In the review of Daniélopolu's book the strength of chlorine solution is given as 0·50 cgm. per 1,000 cc. A reference to the original leaves no doubt that the strength was

0·5 gm. per 1,000 cc. The confusion arose from the common Continental habit of writing 0·50 cgm., meaning 50 cgm. or 0·5 gm.]

John W. D. Megaw.

SAUTER, H. B. Ueber die Nachprüfung und Auswertung einer Schnell-Agglutination bei Fleckfieber. [**Confirmatory Trials of a Rapid Agglutination Test in Typhus Fever.**] *Deut. med. Woch.* 1944, Feb. 4, v. 70, No. 5/6, 63–4, 7 figs.

BOHNENKAMP. Bemerkungen zu der vorstehenden Arbeit von Oberarzt Dr. H. Sauter. [**Comments on the Foregoing Work.**] *Ibid.* 64.

The rapid-agglutination reaction mentioned in the preceding abstract was tested by the author, whose article is illustrated by seven clearly reproduced photographs showing the various types of flocculation that occurred.

It was found that readings of the reaction could be made within one minute when 30-hour broth cultures of *Proteus OX19* were used for seeding the agar plates from which suspensions were prepared, as contrasted with a period of three to four minutes needed when 18-hour cultures were employed.

Flocculation was found to be less satisfactory when sodium citrate was mixed with the blood under test.

With blood whose Weil-Felix titre was less than 1–600 the size of the floccules varied in proportion to the titre, but when the titre was higher no further change was observed.

Among the 10 cases tested the earliest positive reaction occurred on the 5th day, and in this case the standard test still gave a negative reaction.

In one person who had been attacked a year previously the reaction was definitely positive although the Weil-Felix titre was only 1–10.

John W. D. Megaw.

BECK, M. Dorthy, BODILY, H. L. & O'DONNELL, Rosemary. **A Strain of Typhus Rickettsiae isolated from the Brain of a Wild Rat in California.** *Pub. Health Rep.* Wash. 1944, June 2, v. 59, No. 22, 701–12, 4 figs. [24 refs.]

For the first time in California a strain of typhus rickettsia has been recovered from the brain of a wild rat, *R. rattus alexandrinus*.

The rat was trapped on a poultry farm in San Bernardino County in March 1943, in a room immediately over a rest room which had been used by a woman who had recently been attacked by typhus fever. The Weil-Felix titre in her case rose to 1–5,120.

The investigation was very thorough, and the present report will be found of great interest to all who are engaged in research on rickettsiae.

Two guineapigs inoculated intraperitoneally with the brain substance of the rat developed fever on the 8th and 11th days, and each animal had scrotal swelling one or two days after the onset. The strain isolated from one of the guineapigs was maintained through 25 serial passages through a total of 69 guineapigs, two of which died of intercurrent infections two and four days after inoculation. All the other animals showed a typical rise in temperature, and 64 of them developed scrotal involvement ; only two died.

When scrotal material was used for inoculation the incubation period was about three days, with blood it was about seven days, and with brain substance about eight days.

Two other rats and 21 mice trapped in the same building were negative; so also were three pools of fleas, including one from the infected rat.

White rats inoculated with the strain in the same way as guineapigs developed inapparent infections; so also did eight young cotton rats, and from the brains of two of these the strain was recovered 24 days after inoculation.

The strain was passaged repeatedly through white mice by intraperitoneal injections of brain substance, and caused typical reactions.

It was cultivated by the yolk-sac method.

Infection with the strain caused complete immunity in guineapigs against standard epidemic and endemic strains of Rickettsiae.

Intracellular rickettsiae were found in smears made from the tunica vaginalis of infected guineapigs, and inoculated rabbits showed a rising-titre agglutination with *Proteus OX19.*

John W. D. Megaw.

SMORODINTZEFF, A. A. & FRADKINA, R. V. **Slide Agglutination Test for Rapid Diagnosis of Pre-Eruptive Typhus Fever.** *Proc. Soc. Exper. Biol. & Med.* 1944, June, v. 56, No. 2, 93–4.

The practical application of this test is based on the observation of DROBYSHEVSKAYA and SMORODINTZEFF (*J. Epid. & Microbiol.*, Moscow, 1942, No. 1) that a specific antigen occurs in the serum of patients during the first few febrile days of typhus fever. About 60 per cent. of 107 typhus patients studied during the pre-eruptive stage had enough antigen to be detected by the complement-fixation test, and the antigen was absent from sera of 57 patients with typhoid, dysentery and pneumonia. The specific antigen could no longer be detected after the sixth to the ninth day when antibodies appeared.

The technique described in the paper was devised as a simple method of demonstrating the presence of the antigen in the early stages of attacks of typhus fever. The results obtained by this simple and rapid slide test were found to be comparable with those of the complement-fixation test.

Chemically-pure carmine is finely ground in a mortar and is made into a 2 per cent. suspension by the gradual addition of distilled water while the carmine is being constantly triturated. After shaking the mixture for 10 minutes in a vessel with glass beads the larger particles are removed by a short period of slow centrifugation. The suspension is then kept for 24 hours, then centrifuged to precipitate most of the carmine particles. After decanting the supernatant fluid the sediment is resuspended in the original volume of 0·25 per cent. solution of NaCl.

Serum to be tested (finger blood, lysed in distilled water, can also be used) is diluted 1 to 5 with distilled water and one-quarter volume of the carmine suspension is added. The mixture is shaken for 5 to 10 minutes, then lightly centrifuged (500 r.p.m. for 5 minutes); most of the supernatant fluid is decanted and the sediment is resuspended in the residual fluid. Such antigen-laden particles of carmine are specifically agglutinated by the human serum rich in antibody, but the intensity of the reaction is increased several-fold if the antibody is used not as such, but also adsorbed on particles of carmine. " In the actual

test, two drops of the suspension of carmine, laden with serum collected during the acute phase of the disease (antigen), are placed separately on a glass slide. To one of them is added a drop of a suspension of carmine particles laden with antibody-containing serum; to the other, as control, is added a suspension of particles to which negative serum was adsorbed." Agglutination becomes visible macroscopically within a few minutes.

Solutions with low concentration of electrolytes must be used; even so, certain lots of carmine are unusable because of non-specific aggregation.

Sera should be used within five days. *John W. D. Megaw.*

VORONINAA, E. V. & MARKOVICH, A. V. **[Castañeda's X-factor in the Diagnosis of Typhus.] (Preliminary Communication.)** *Zhurnal Mikrobiologii, Epidemiologii i Immunibiologii.* Moscow. 1943, No. 12, 25–7. [12 refs.] [In Russian.]

The aim of the work described in this article was to isolate the X-factor from the culture of *Proteus X19*, in order to use it as a diagnostic factor in typhus by the precipitation of this polysaccharide with the serum of a typhus patient. This precipitation reaction would be simpler and more accessible than the Weil-Felix reaction, as it could be performed under primitive conditions. The method of CASTAÑEDA, which consists of a dissolution of an agar culture of *Proteus X19* by antiformin with subsequent sedimentation with alcohol, revealed an alkali-stable X-fraction [see *Bulletin of Hygiene*, 1934, v. 9, 776]. With this fraction a ring-precipitation test was done with different sera. The results were as follows:—

Serum	Kind of animal	Titre of agglutination of *Proteus X19*	Result of precipitin reaction with X-factor of Castañeda.
H2426 typhus	Man	1 : 800	++++
H59 ,, ...	Man	1 : 800	++++
H57 ,, ...	Man	1 : 800	++++
H53 ,, ...	Man	1 : 800	++++
Convalescent from measles ...	Man		0
Rabbit immunized by *Proteus X19*	Rabbit	1 : 5120	++++
Antipneumococcal	Rabbit		0
Antimeningococcal	Rabbit; horse		0

This table shows that a positive precipitin reaction can be obtained only with sera of typhus patients and with the serum of a rabbit immunized with a culture of *Proteus X19*. More work on this subject is needed. If this reaction justifies itself it may replace the Weil-Felix reaction, as a much simpler method. *H. W. Swann.*

PALATUCCI, O. A. & MARANGONI, B. A. **Rocky Mountain Spotted Fever.** *Bull. U.S. Army Med. Dept.* 1944, Aug., No. 79, 116–20, 2 figs.

Three cases of Rocky Mountain spotted fever are described; two of these were "proved" and one was "inconclusive." They occurred in

Alabama in 1943, and in the two cases regarded as proved the complement-fixation test gave great help in diagnosis. In both of them this reaction was strongly positive; the Weil-Felix reaction with *Proteus OX19* was also strongly positive; with *OX2* it was negative in one case and weakly positive in the other. There was no history of tick-bite in either case.

In the inconclusive case there was a history of tick-bite; *OX19* was agglutinated at a titre of 1–320, and *OX2* at 1–160; the complement-fixation reaction was positive, 1–16 with Rocky Mountain spotted fever antigen, and 1–4 with murine antigen.

The total leucocyte count in one of the proved cases was 3,600 on the eighth day. *John W. D. Megaw.*

PLOTZ, H., WERTMAN, K. & REAGAN, R. L. **Laboratory Aids in Diagnosis of Rocky Mountain Spotted Fever.** *Bull. U.S. Army Med. Dept.* 1944, Aug., No. 79, 40–44.

The authors point out that difficulties in differential diagnosis are likely to occur in places where two or more of the fevers of the typhus group occur, for example " in North Africa where epidemic and murine typhus and *fièvre boutonneuse*—a disease of the Rocky Mountain spotted fever group—may exist in the same region."

Type	Day of disease	Complement-Fixation Titre*			Weil-Felix Titre†	
		R.M. spotted fever	Epi-demic	Murine	*Pr. OX19*	*Pr. OX2*
Murine	8	0	0	0	40	0
	31	0	0	320	640	0
Murine	4	0	0	0	0	0
	19	0	0	40	640	80
	36	0	0	320	320	40
Murine	12	0	0	0	1,280	80
	23	0	0	160	5,120	0
Epidemic	8	0	0	0	0	0
	12	0	40	0	640	40
R.M. spotted fever ...	7	0	0	0	40	80
	11	160	0	0	80	1,280
R.M. spotted fever ...	5	0	0	0	0	0
	12	80	0	0	640	0
R.M. spotted fever ...	7	0	0	0	160	0
	14	160	0	0	640	0
R.M. spotted fever ...	13	0	0	0	0	0
	24	160	0	0	320	0
R.M. spotted fever (fatal)	13	0	0	0	0	0
	16	40	0	0	0	0

* Initial serum dilution. † Final serum dilution.

The help given by the complement-fixation test is illustrated by nine examples of the responses in sera examined at the Army Medical School, Washington, where the test is now carried out regularly. About 5 cc. of serum collected in conditions ensuring sterility are needed.

The authors state that although a high-titre *OX2* agglutination is suggestive of Rocky Mountain spotted fever the reaction with this organism may be weak or even negative, and that the reaction with *OXK* may be completely negative in cases of " scrub typhus." They also state that a rising-titre reaction with *OXK* is commonly obtained in scrub typhus, and is not found in any other known disease.

John W. D. Megaw.

THEILER, G. **Notes on the Ticks off Domestic Stock from Portuguese East Africa.** 55 pp., 49 figs. [36 refs.] 1943. Estação Anti-Malárica de Lourenço Marques.

Contains notes on *Amblyomma hebraeum*, *Hyalomma impressum rufipes* (*H. aegyptium impressum*), *Haemaphysalis leachi* and *Rhipicephalus sanguineus*, all concerned in tick-borne typhus, as well as other animal ticks.

HESSE, E. & KREMSER, H. Röntgentherapie chronischer Verlaufsformen des wolhynischen Fiebers. [**The X-Ray Treatment of Chronic Types of Trench Fever.**] *Deut. med. Woch.* 1944, Feb. 18, v. 70, No. 7/8, 96–7, 2 charts. [16 refs.]

X-ray treatment was employed in 49 cases of trench fever.

The dosage is stated to be 110 r. (100 k.v.; 6 m.a.; 3 mm. aluminium filter; tube distance 40 cm.) For the head region 180 k.v. and a copper filter of 0·5 mm. were used.

The first application is made to the lumbar and lower dorsal region of the spine over an area of 10 × 55 cm. Two days later the shin bones, and sometimes also the knee and ankle joints, are irradiated. Again, after two days, the application is made to the area first treated. Later treatment depends on the localization of the pains.

For severe headache the temporal region is irradiated on both sides, at intervals of three days, up to a maximum of three applications. Epilation is avoided.

Four examples are given to show modifications in the sites of application and the effect of the treatment.

In acute attacks the temperature is said to fall to normal within 3 to 11 days (average 6); in chronic cases the response is slower, but the pains are relieved after a single sitting and are usually cured after four or five applications.

In four cases the temperature rose higher during the treatment, and in one a subfebrile temperature persisted for several weeks after complete freedom from pain had been produced.

The treatment should be restricted to cases in which the services of skilled physicians and technical experts are available.

[No reference is made to the employment of controls, though the great variability of the disease is admitted. Also, the possible psychological effect of the treatment on the pains is not mentioned.]

John W. D. Megaw.

ERRATUM.

In the abstract of J. C. PATEL's paper on " Typhus Fever in Bombay " in this *Bulletin*, 1944, Feb., v. 41, No. 2, at p. 118, and in a reference to

this paper in the review article on " The Distribution of Mite-Borne Typhus Fever " in this *Bulletin*, 1944, June, v. 41, No. 6, at p. 437, it was wrongly stated that the serum of one of Patel's cases in Bombay was positive to *OXK* at a 1/1,500 dilution. This should read 1/500.

YELLOW FEVER.

LIEGEOIS, P. Fièvre jaune au Congo Belge. [**Yellow Fever in the Belgian Congo.**] *Rec. Travaux Sci. Méd. Congo Belge.* Léopoldville. 1944, Jan., No. 2, 97–123, 1 map. [11 refs.]

A general account of the prevalence of yellow fever in the Belgian Congo with special reference to observations extending from 1938 to the beginning of 1942 when this article was written.

After reviewing earlier records of the disease and the work of the Rockefeller Commission in 1932 and 1933, the author describes the methods subsequently used in the study of the problem and also the results obtained in recent years.

Viscerotomy has been used, especially since 1938, for the collection of liver samples from six provinces. A total of 4,340 specimens from Stanleyville were examined during 1939, 1940 and 1941, and 348 from other provinces. Yellow fever was diagnosed in two cases from Stanleyville, one from Coquilhatville, and two from Léopoldville, in addition to another case from Bondo diagnosed previously. It is of interest that all these six localities are widely separated and in the interior of the country.

Each case is considered in detail and mouse protection tests were made with the blood of children and adults in the neighbourhood. In no instance was it possible to find any other cases of yellow fever in the vicinity.

The results of protection tests carried out with blood samples collected in various provinces are given in tabular form. The country is divided into three regions :—A, comprising territories in which the percentage of positive results in adults is relatively high, and also sera from children are often protective ; B, territories in which adults give a certain number of positives, but children are only rarely positive ; and C, territories in which positives have not been recorded.

In region A, comprising the territories of Bondo, Ango, Niangara and Dungu, out of 353 adults 26·6 per cent. were positive and of 881 children (up to 14 years old) 8·7 per cent. were positive.

In region B, comprising the territories of Alceti, Buta, Poko, Paulis, Faradje, Banalia, Stanleyville, Bafwasende, Irumu, Ponthierville, Opala, Isangi (Lubutu), the comparable figures were adults 654, with 6·8 per cent. positive, and children 458, with 0·7 per cent. positive, doubtful results being excluded.

Details are given of entomological studies carried out in the Province of Stanleyville, including a list of the species of Culicine larvae collected. During 1939 and 1940, *Aëdes aegypti* was found breeding in 4,389 specified sites, and the localities in which these mosquitoes occurred confirm the view that in the Belgian Congo this species is more common in European quarters than in the neighbouring African towns and villages.

The author discusses the nature of the six recorded cases of yellow fever and is of the opinion that, in spite of the presence of *Aëdes aegypti*, these were probably cases of jungle yellow fever. Also it is difficult to explain their occurrence except by the assumption of some reservoir of infection other than man. *E. Hindle.*

SAWYER, W. A., MEYER, K. F., EATON, M. D., BAUER, J. H., PUTNAM, P. & SCHWENTKER, F. F. **Jaundice in Army Personnel in the Western Region of the United States and its relation to Vaccination against Yellow Fever. Parts I, II, III & IV.** *Amer. J. Hyg.* 1944, May, v. 39, No. 3, 337–430 and July, v. 40, No. 1, 35–107, 17 figs. [70 refs.]

In March 1942 jaundice appeared in unusual incidence in U.S. Army troops in California and large numbers of cases continued to occur into December 1942 in this region and elsewhere. In Part I the authors present the statistical data gathered from persons inoculated with yellow fever vaccine in the western region of the U.S.A. An analysis of 10,284 cases of jaundice showed that most of the cases were related to specific lots of vaccine. Geographical location of the affected units was not an actiological factor. The mean intervals between vaccination and onset of jaundice remained almost constant no matter where or when incriminated lots of vaccine were inoculated and is evidence of the close association of the two events.

There had been a few small outbreaks of jaundice among civilians in California prior to the onset of jaundice in military units but the authors do not believe that these could have given rise to the widespread epidemics in the Army.

In Part II there is first a description in detail of the development of vaccination against yellow fever, the methods and material used in the production of the vaccine, and complications that have occurred previously following injection of certain lots of the vaccine made in England and Brazil. This is followed by a discussion of the various lots of vaccine which produced jaundice in the U.S. Army as described in Part I and the possible sources of the icterogenic agent present in the vaccine. The remainder of the article is concerned with attempts to transmit the disease to experimental animals and serological studies. The description of the development of vaccination and present methods of production is too detailed to be abstracted but should be read by all medical officers likely to come in contact with this material so that they will appreciate the special character of this vaccine.

The complications of jaundice in England and jaundice and encephalitis in Brazil have been described already and by others.

In Part I it had been shown that jaundice had followed the inoculation of certain batches of the vaccine no matter where or when it was given. An examination of the distribution of 117 lots issued to the Army showed that 98 lots had been associated with 26,771 cases of jaundice. The authors do not consider that this includes all cases that occurred as many were not reported and it is not known how many doses of each lot were actually used or exactly how many cases occurred with each lot. The number of cases of jaundice resulting from a single lot varied between 1 and 5,360 and the case rate per 1,000 doses net issue between 0·04 and 95·8. However, the great majority of the cases appeared to be related to only seven lots and an attempt was made to divide the vaccine lots into different categories according to their icterogenic

property. Because of the number of presumed unreported cases and the wastage of vaccine it was considered that all case rates per 1,000 doses of vaccine should be multiplied by at least a factor of 2. Forty-seven of the 117 lots were found to have a case rate greater than between 1 and 2 per 1,000 per annum, the normal incidence of jaundice in the U.S. Army for the past 10 years. Nine of the 47 lots had a case rate between 10 and 100 per 1,000 doses net issue, and 38 lots a case rate of 1 to 10 which, when multiplied by the factor of 2, have a rate higher than the normal rate in the Army. There seems to have been an extremely large variability in the case rates with different batches and even with the same batch in different localities, *e.g.*, 64,300 doses of lot 331 were issued to the Army and produced 1,750 cases, or 27·22 per 1,000 doses, whereas 1,039 doses of the vaccine inoculated into civilians in the Virgin Islands resulted in 153 cases, or 147 per 1,000 doses. [It is interesting to note the comparatively small incidence in these groups compared with that in recent reports of jaundice following the use of yellow fever vaccine, pooled "normal" serum and pooled convalescent mumps plasma in England where figures of between 30 and 57 per cent. have been recorded.

In recent years the general assumption has been that the delayed jaundice which has followed the inoculation of certain batches of yellow fever vaccine has been the result of the introduction of an icterogenic agent present in the supposedly normal human serum used to make the vaccine. The proof of the correctness of this hypothesis was recently provided by MacCallum and Bauer who were able to test some of the serum which had been used to make an icterogenic batch of vaccine. When injected subcutaneously this serum produced the same proportion of cases of jaundice as the vaccine which had contained it. The assumption has usually been that some donor has been suffering from infective hepatitis in an unrecognized form and that the hypothetical virus of this disease has been circulating in the blood at the time the blood was collected. However, in all previous instances of so-called homologous serum jaundice, in some of which all the donors who contributed to the incriminated material have been traced, there has been a complete failure to find a suspicious donor.]

In the present incident 970 donors had been bled to provide 311 litres of human serum for 141 lots of human vaccine between December 20th, 1940, and March 18th, 1942. When 367 of the donors were interviewed it was found that 23 of them had a history of jaundice with the time of attack varying from early infancy to a few weeks before bleeding in one instance; six of the 23 donors were bled twice, one three times and one four times. Each of the 23 donors contributed to an icterogenic lot of vaccine although not on each bleeding. Donors with a history of a relatively recent attack of jaundice contributed serum to the weekly shipment which was used in the preparation of most of the highly icterogenic lots but no previously jaundiced donor contributed to a number of the moderately icterogenic lots. One donor, a physician, provided blood 52 days after the appearance of jaundice, when his sclerae were still icteric! His serum formed part of pools used for making one highly icterogenic and four moderately icterogenic lots of vaccine. If these 23 donors are to be incriminated then the icterogenic agent must have been present in their blood intermittently at intervals of 52 days to many years after the onset of their jaundice. Conditions somewhat similar are known to occur in certain virus diseases, *e.g.* infectious anaemia of horses where virus has been shown to be present

many years after recovery from an attack. The small amount of experimental evidence available does not suggest that this is the case in men, though more data are obviously necessary to settle this point [OLIPHANT, GILLIAM and LARSON (*Pub. Health Rep.*, 1943, v. 58, 1233) failed to find an icterogenic agent in the serum of one individual 2½ months after jaundice had subsided and MacCallum and Bauer obtained similar negative results with serum collected from a single individual 66 and 141 days after the first appearance of jaundice].

The possibility that the seed virus or the chick embryos used for making the vaccine were contaminated is shown to have been practically impossible and the hepatitis bore no relation to yellow fever. All yellow fever vaccine has been free from serum for two years or more now and there should be no recurrence of such incidents. All attempts to transmit the disease to experimental animals have been unsuccessful except in the case of the horse ; three of five horses in one group and two of four in another developed an increase in serum bilirubin several months after inoculation. Further experiments are being carried out and will be reported later.

Serological studies showed that an antigen appeared, in the acute stage of the disease, which might react with an antibody present in the convalescent phase. The reactions were relatively weak and rather difficult to demonstrate. They were not specific for the disease but were similar to those described previously by HUGHES [this *Bulletin*, 1934, v. 31, 83] for yellow fever. Positive results were also obtained with sera from cases of dengue with jaundice. Further studies revealed that this antigen-antibody system was derived from the liver and was organ-specific, regardless of the animal species from which the liver originates. The authors concluded that the reaction cannot be used for diagnostic purposes either in yellow fever or post-vaccination hepatitis. Further studies of this reaction have recently been reported by EATON and his colleagues [see *Bulletin of Hygiene*, 1944, v. 19, 746].

F. O. MacCallum.

FINDLAY, G. M., MARTIN, N. H. & MITCHELL, J. B. **Hepatitis after Yellow Fever Inoculation. Relation to Infective Hepatitis.** *Lancet.* 1944, Sept. 2, 9 & 16, 301–7 ; 340–44 ; 365–70 ; 4 figs. [44 refs.]

In Part 1 of this communication the authors describe the clinical and pathological findings in cases of jaundice in West Africa theatre arising in persons who had previously received yellow fever vaccine. In 670 cases between December 1942 and July 1943 the latent period from inoculation to onset of jaundice ranged from 26 to 239 days with an average of 101·5 days. The clinical manifestations were analysed in 432 cases. The length of the pre-icteric stage varied from 0 to 56 days with an average of 7 days, and was independent of the interval since inoculation. The common symptoms in order of frequency were anorexia, nausea, epigastric pain, bad taste in the mouth, vomiting, lassitude, constipation and headache, and these symptoms most frequently appeared on the first day of the disease. Joint pains occurred in 5·5 per cent. of cases. Jaundice and biliuria were present in all cases, the liver was enlarged in 55·5 per cent. and the spleen in 48·6 per cent. [Text and tables differ on this and other points, the text giving a figure of 13·6 per cent.] Skin rashes, chiefly urticarial,

were present in 4·6 per cent. Fever was observed in 17·6 per cent. after the onset of jaundice and usually lasted three to four days; in 11 cases there was persistent fever for two to three weeks. Haemorrhagic manifestations included skin purpura (five cases), haematuria (four), haematemesis (four), epistaxis (one) and conjunctival haemorrhage (one). A search for sub-icteric cases in one unit revealed three out of 41 cases with biliuria but no jaundice; four other men had symptoms suggestive of infective hepatitis without jaundice and biliuria. Relapses occurred in 14 instances (2 per cent.), the interval after discharge from hospital ranging from 2 to 61 days. In three cases a relapse appeared to have been precipitated by an attack of malaria. Histological findings are described in the one fatal case in the series but unfortunately the hepatitis was associated with malignant tertian malaria. Malarial parasites were found in the blood of 18 (3·8 per cent.) of 465 patients examined, so that in these it was not possible to exclude malaria as the cause of the hepatitis. On the basis of their clinical and pathological findings the authors conclude that the disease is indistinguishable from naturally occurring infective hepatitis.

In Part 2 the authors present epidemiological and immunological studies. It is suggested that secondary cases arose in contacts of cases of post-inoculation jaundice. In support of this opinion it is stated that notifications of infective hepatitis increased during the period of the epidemic amongst vaccinated persons; moreover, in individual units cases of infective hepatitis appeared amongst new arrivals and at irregular rather than monthly intervals, from which it is inferred that the infection was contracted from post-inoculation cases occurring at the time. A history of a previous attack of jaundice was given by one officer (1·1 per cent.) and three other ranks (0·5 per cent.). These figures were found to be significantly smaller than the previous jaundice rates for an unselected section of the community, whence it is argued that infective hepatitis confers immunity against yellow fever inoculation jaundice. A summary is given of complement fixation tests carried out, in which the antigen was an extract of liver from a fatal case of hepatitis. Five out of six sera from patients recovered from post-inoculation jaundice are stated to have given positive fixation, but were negative with control antigen. It is claimed that these results also demonstrate the identity of infective hepatitis and post-inoculation jaundice. Finally, an account is given of unsuccessful attempts to transmit the disease to animals and human volunteers.

[Many of the authors' conclusions would be valid only if it were quite certain that, in the cases described, the hepatitis was attributable to yellow fever vaccine. This is particularly true of the evidence for secondary spread to contacts. Since infective hepatitis was prevalent at the time of the outbreak, the diagnosis of post-inoculation jaundice rests on the finding of significantly different attack rates following different batches of vaccine used over the same period. The argument would be more convincing if details had been given and if the batch numbers of the icterogenic vaccine had been stated. Even so, in the presence of infective hepatitis the diagnosis of post-inoculation jaundice could not be made with certainty in individual cases. It is notable that the outbreak which the authors attribute to yellow fever vaccine occurred about the same time as the epidemic of infective hepatitis in the Middle East.] *C. Wilson.*

KOPROWSKI, H. & LENNETTE, E. H. **Propagation of Yellow Fever Virus in Tissue Cultures containing Sulfonamides.** *Amer. J. Hyg.* 1944, July, v. 40, No. 1, 1–13, 1 fig. [19 refs.]

The authors have failed to inhibit the propagation of the 17DD High substrain of yellow fever virus in tissue cultures of minced chick embryo and serum tyrode by the addition of maximal concentrations of sulphapyridine or sulphathiazole. In order to overcome any slight inhibitory effect of the minced chick tissue on the drug, the tissue culture medium contained 20×10^{-4}M concentration of the drug.

Neither multiplication of the virus nor accumulation of metabolites and tissue breakdown products inhibited the activity of the drug.

The authors suggest that the addition of the drugs provides a simple and effective means of protecting virus cultures against bacterial contamination and should prove of value in attempts at direct isolation in the field of virus from contaminated sources. *F. O. MacCallum.*

KOPROWSKI, H. & LENNETTE, E. H. **Sulfonamides in Yellow Fever Virus Infections of Mice and developing Chick Embryos.** *Amer. J. Hyg.* 1944, July, v. 40, No. 1, 14–25. [35 refs.]

In 1938 FINDLAY and MACCALLUM noted briefly that " yellow fever infections in monkey and mouse have been unaffected by prontosil and allied drugs." The authors have obtained similar negative results in adult mice injected intracerebrally and baby mice injected extraneurally with the French neurotropic strain of yellow fever virus and treated with sulphapyridine or sulphathiazole in amounts approaching the maximum tolerated dose.

They also injected 5–15 mgm. of sodium sulphapyridine daily for four successive days on to the chorio-allantoic membrane of chick embryos which were infected with the French neurotropic strain either shortly before or shortly after the first dose of drug. The drug was unable to prevent multiplication of the virus in eggs inoculated with as little as 10 intracerebral 50 per cent. mouse lethal doses.

F. O. MacCallum.

DENGUE AND SANDFLY FEVER.

LEROY, G. V. & LINDBERG, H. A. **The Diagnosis of Dengue.** *Bull. U.S. Army Med. Dept.* 1944, Aug., No. 79, 92–100, 2 figs. & 1 map.

A description is given of the clinical features of 71 closely-observed cases of dengue among U.S.A. soldiers aged 26–35 years. The place of occurrence is not stated.

Break-bone pains occurred in only a few of the older patients. Progressive enlargement of the lymph nodes, with tenderness on firm pressure, was a striking feature; the posterior cervical, submental, and post-auricular glands were most often involved.

The average duration of the fever was six days, the range was three to eight days.

The majority of the patients had a rash.

The fever curve showed an abrupt rise to 102°–104°F.; after 24 to 36 hours the temperature fell to 99°–102°F. and kept at the same level

for two to four days ; then there was a terminal rise to a higher level than the initial temperature, followed by an abrupt fall to normal after one to two days. The temperature seldom fell to normal in the period before the terminal rise.

The blood changes were confined to the leucocytes ; the average picture was as shown in the table :—

Day of disease	Granulocytes (per cent.)	Lymphocytes (per cent.)	Total count
1st	72	19	6,128
4th	58	32	5,174
7th	42	47	
8th	33	54	6,200
10th	45	47	6,700
12th	57	43	6,700

The average of the lowest leucocyte counts for each patient was 4,630 ; the range of the lowest counts was 2,100–7,400.

The pathognomonic blood picture is stated as being :—(1) Moderate leucopenia, most marked during the terminal rise of temperature. (2) The presence of vacuolated monocytes throughout the fever. [In another place the authors state that these cells " are not truly pathognomonic."] (3) The development of marked neutropenia and lymphocytosis during convalescence. [Elsewhere the authors say that " it is difficult to state whether an absolute lymphocytosis occurred or not," so presumably they refer to a relative lymphocytosis.] (4) The presence of Türk's irritation leucocytes during convalescence. They add that " the marked leucopenia in convalescence is presented as a specific diagnostic feature of dengue." [The authors, perhaps unintentionally, convey the impression that they regard the type of fever curve and the blood picture described by them as diagnostic features of universal applicability in dengue.] *John W. D. Megaw.*

Sabin, A. B., Philip, C. B. & Paul, J. R. **Phlebotomus (Pappataci or Sandfly) Fever: A Disease of Military Importance. Summary of existing Knowledge and Preliminary Report of Original Investigations.** *J. Amer. Med. Ass.* 1944, July 1 & 8, v. 125, Nos. 9 & 10, 603–6 ; 693–9, 9 figs. [Refs. in footnotes.]

The authors of this paper had been members of a Commission which studied sandfly fever in North Africa in 1943. The findings are based chiefly on the investigation of more than 100 cases of the disease produced experimentally in volunteers belonging to the U.S. Army.

Serum obtained from naturally-infected soldiers in the Middle East and Sicily was the original source of the infecting material.

The virus was present in the blood at least 24 hours before the onset and during the first 24 hours of the fever, but never later than 48 hours after the onset. It was passaged in series seven times from man to man without obvious change in its properties.

About 95 per cent. of the volunteers were susceptible to intracutaneous or intravenous inoculation, but similar doses of infective serum failed to produce the disease in 50 to 75 per cent. of the same class of subjects when the injections were administered by the subcutaneous or intramuscular routes.

Each cc. of infective serum obtained at the onset contained about 1,000 infecting doses—probably sometimes less. No virus could be demonstrated in the cerebrospinal fluid.

By gradocol-membrane filtration tests the range of size of the virus was estimated to be as low as 25–37 millimicrons, so it was thought to be nearly as small as that of the yellow-fever virus.

Young baboons and several species of monkeys (*Cercopithecus griseoviridis*, *C. aethiops centralis*, *C.* [*Erythrocebus*] *patas*, *Macaca radiata* and *M. mulatta*) and rodents (mice, hamsters, jerboas, rabbits, guinea-pigs and cotton rats) gave negative responses to inoculation.

The virus was not found capable of producing specific plaques on the chorio-allantoic membranes of chick embryos, and negative results followed inoculation of the virus into any part of these embryos. So also there was no evidence of the presence of the virus in embryos after attempts to transmit infection through them by three or four passages carried out according to the technique used in work on the yellow-fever virus.

Laboratory-reared *Phlebotomus papatasi* transmitted infection from patients to healthy persons, though there was one failure in the case of a person bitten by many infected sandflies, in spite of the fact that he was later shown to be susceptible.

Control tests with *Culex pipiens*, *Aëdes aegypti* and *Pulex irritans* gave negative results. Two persons who were later found to be susceptible were bitten by 93 and 195 *Aëdes aegypti* mosquitoes which had fed one or more times on infected patients; the results were negative in both cases.

P. papatasi larvae were fed on infected human serum; these in the adult stage were not infective. So also sandflies whose parents had been proved to be infective failed to produce the disease in susceptible persons. These experiments were not extensive and thus were not regarded as invalidating the findings of Whittingham [this *Bulletin*, 1925, v. 22, 123] and others (Moshkovsky *et al.*, 1937, *Med. Parasit. & Parasitic Dis.*, Moscow, v. 6, 922) who have shown that the offspring of infected sandflies are sometimes infective.

The description of the clinical features of the disease is based on more than 100 experimental cases. In 85 per cent. of the subjects the duration of the fever was two to four days; the extreme range was from a few hours to nine days. A scarlet flush of the face and neck was often seen, and, apart from this, varying types of rash were observed in 5–10 per cent. of the cases. The authors mention that Franz (Doerr, Franz and Tassig, "Das Pappatacifieber," Leipzig, Franz Deuticke, 1909) recorded a rash in 30 per cent. of his cases.

Leucopenia (less than 5,000 white cells per cmm.) occurred after the first day in 90 per cent. of the cases, but greater help was obtained in diagnosis by daily observations of the total and relative counts of the mature (segmented) and immature ("staff") neutrophiles. After the first day the former rapidly diminished in numbers and the latter increased to a striking degree. This was a constant phenomenon to which sufficient attention had not hitherto been given. [It has been described as a feature of dengue.]

Attempts to find a specific test failed; they included efforts to elicit a skin test and complement-fixation and precipitin reactions.

Immunity was proved to exist in eight persons four to six weeks after recovery, and in 10 others after four months. Two medical men who gave histories of natural attacks four and seven years previously were

tested; one had a typical, and the other an abortive, attack after inoculation with the virus.

Attempts were made to produce immunity without causing the disease; in one of the experiments 11 persons were inoculated intracutaneously with virus inactivated by ultra-violet irradiation, and although they gave no response they were afterwards found to have been immunized.

The authors emphasize the well-known fact that many cases of the disease are diagnosed and treated as malaria because proper blood examination has not been carried out.

Applications of dimethyl phthalate or of a pyrethrum vanishing cream were found to give protection against sandfly bites for six to eight hours.

In addition to the above and other findings by the Commission the paper contains a comprehensive review of existing knowledge of sandfly fever, which has become an important war disease.

John W. D. Megaw.

Cottrell, J. D., Stronach, R. D. & Peddie, J. J. G. **A Syndrome of Generalized Lymphadenitis with Neutrophil Leucopenia.** *J. Roy. Army Med. Corps.* 1944, July, v. 83, No. 1, 12–16.

An outbreak of a problematic type of short fever occurred in Tripolitania from May 1943 to an unspecified later date. There were 40 cases, of which 25 were closely observed, and in several of the latter the patients were members of the medical and nursing staff of a New Zealand Military Hospital.

The disease ran a short febrile course "like a mild sandfly fever." There was a more or less generalized enlargement and tenderness of the lymphatic glands, starting within the first 48 hours and lasting 5–15 days. The size of the glands ranged from a millimetre or so in diameter to 1·5 or even 2·5 cm. [presumably in length]. In most cases there was at least one group of larger size, sometimes associated with a local septic condition such as desert sore. The posterior cervical glands were the first to enlarge in most cases, but any or all of the superficial gland groups might be affected, sometimes only on one side.

The average duration of the fever was five days, the maximum was seven days. The temperature seldom exceeded 102°F., but occasionally reached 104°. There was frontal headache, retro-ocular pain, and mild aching of the back and limbs. In one case the fever recurred for two days after an afebrile period of two days.

A constant feature was stiffness of the neck, and often there was redness of the pharyngeal mucous membrane. One patient had an ill-marked erythematous rash on the back and chest from the eighth to the tenth day.

The neutrophil leucocytes usually fell progressively in number to 315–2,470 per c.mm., and for an average of 3·8 days they were less than 45 per cent. of the total count. In one case the neutrophils fell to 16 per cent., and the lymphocytes rose to 73 per cent., but there was seldom any increase in the total number of the lymphocytes. A cell somewhat resembling a Türk's irritation cell was seen.

The mode of spread was quite unknown. In an officers' mess with a personnel of 25 there were five cases which occurred on the following days of June: 11th, 12th, 20th, 23rd, 25th and 27th. All but three of the 40 patients had been stationed within 15 miles of Tripoli for some time before the onset.

During the course of the "epidemic" a few sporadic cases resembling sandfly fever were treated at the hospital, and in two or three other cases the features were similar to those described above except that there was no pronounced neutropenia.

The authors suggest that the disease may be an unusual type of sandfly fever or may be due to an unknown virus of the sandfly-dengue group; they state that "the features were not those of dengue." They do not absolutely exclude a disease related to infective mononucleosis in spite of the fact that the Paul-Bunnell test was negative.

[Information about the types of fever curve and the prevalence of possible insect vectors is lacking, but the features described seem to justify the authors' suggestion that the disease was caused by a virus belonging to the dengue-sandfly fever group.] *John W. D. Megaw.*

PLAGUE.

Hecht, O. Consideraciones entomologicas a la epidemiologia de la peste bubonica de los roedores en Venezuela. [**Entomology in Rodent Plague of Venezuela.**] *Rev. Sanidad y Asistencia Social.* Caracas. 1943, v. 8, No. 6, 1159–62. English summary (5 lines).

A previous communication by the author showed [this *Bulletin* 1943, v. 40, 605] that in a sparsely populated region of the State of Aragua in Venezuela the examination of sylvatic rodent fleas furnished 97·2 per cent. of *Rhopalopsyllus* fleas. Now the experiments of Eskey and Haas [this *Bulletin*, 1939, v. 36, 968] have demonstrated that *Xenopsylla cheopis* is the most efficient flea transmitter of epizootic plague, but that quite a number of other species can transmit plague and can become "blocked" fleas. Moreover the fleas of sylvatic rodents have an important peculiarity in that they can become infected with plague, survive for two to four months and then can become dangerous blocked fleas. On the strength of this argument the author contends that the small epidemic plague (19 human cases) in July-August 1943 could, by analogy with the experimental results, be related firstly, for the human cases, to *Xenopsylla brasiliensis*, secondly, to their appearance not far from a sylvatic plague region south of Tejerias, and thirdly to the appearance of *Xenopsylla brasiliensis* among the much larger number of *Rhopalopsyllus* fleas. These were the characteristic features of the flea collection in "No. 1 Zone" in June 1943. It is deduced also that note should be taken in field studies not merely of a flea index but of the species of flea which is prevalent, or again its mixture with known plague vector fleas—in this case of *Rhopalopsyllus* with *Xenopsylla brasiliensis.* *W. F. Harvey.*

Jawetz, E. & Meyer, K. F. **Studies on Plague Immunity in Experimental Animals. I. Protective and Antitoxic Antibodies in the Serum of Actively Immunized Animals.** *J. Immunology.* 1944, July, v. 49, No. 1, 1–14, 1 chart. [28 refs.]

A distinction is always drawn by workers between protective and antitoxic immunization. Such a distinction is particularly liable to be made in the case of antiplague serum whose value is still much disputed.

The authors of this paper evidently consider that " hyperimmune plague antiserum in combination with chemotherapy is the treatment of choice, but its use for prophylaxis is to be discouraged." Their aim has been to obtain a satisfactory test for the measurement of serum potency. Hyperimmune plague sera were obtained from rabbits and a titre was attained which it seemed impossible to transcend by any other method. In order to get this testing serum the rabbits were immunized intravenously by three injections of living avirulent *P. pestis* on alternate days. They were then found to possess serum at its maximum in content of protective antibodies and this already " within a week after the last antigen administration." No correlation was found to exist between the antitoxic and the antibacterial or protective power of a serum. " It is certain, however, that a detectable level of antitoxic antibodies is attained in rabbits only if a re-stimulating injection is given three to four weeks after the primary series of injections of antigen." The authors have developed a mouse test for assaying the anti-infectious and antitoxic antibodies of sera of actively immunized experimental animals. The mice " were injected intra-abdominally with 0·5 ml. of serum dilutions followed 60 minutes later by the intra-abdominal administration of 0·1 ml. of a 10^{-4} dilution of culture. Controls were given 0·5 ml. of normal serum from the same animal species . . ., others 0·5 ml. buffered saline solution followed 60 minutes later by 0·1 ml. of a 10^{-4} or 10^{-5} dilution of culture. The mice were observed for a period of 26 days. . . ." Precise details are given of the mode of elaboration of standards. A detailed discussion of immunity factors, together with abundant references to literature, is presented. *W. F. Harvey.*

Jawetz, E. & Meyer, K. F. **Studies on Plague Immunity in Experimental Animals. II. Some Factors of the Immunity Mechanism in Bubonic Plague.** *J. Immunology.* 1944, July, v. 49, No. 1, 15–30, 1 chart. [32 refs.]

Plague antiserum is a weak serum by comparison with other antisera and the observations here recorded relate mainly, under artificial conditions, to factors of antibacterial immunity in general. Much of the importance of these factors " in the natural mechanism can only be inferred." Conclusions are mostly expressed in negative phraseology, but one positive feature emerges which is that immune animals appear to be able to fix the invading organisms locally. One of the main deductions in this thesis emphasizes " opsonic activity " in contradistinction to a solely " cellular " or " humoral " immunity. Some of the chief conclusions are :— " (1) The serum plasma, or other body fluids of animals immune to plague infection are unable to destroy or lyse *P. pestis* organisms *in vitro* and *in vivo* in the absence of phagocytic cells. (2) Whole blood of plague-immune animals can destroy a much greater number of plague bacilli than blood of normal animals. This higher specific activity is primarily inherent in the plasma, not in the cells. (3) The phagocytic activity of normal serum-cell mixtures does not differ greatly from that of immune mixtures. Only mixtures of immune serum with immune cells, however, are able to produce significant destruction of bacilli. (4) Immune animals are able to fix the bulk of injected *P. pestis* at the site of injection. The mechanism of fixation does not involve lymphatic blockage and deposition of fibrin network to a significant degree. (5) Splenectomy

does not reduce the resistance of immune animals to plague infection. . . . (6) Extracts from both virulent and avirulent plague strains contain a factor which enhances spreading and capillary permeability. . . ." *W. F. Harvey.*

GATES, Doris B. *Xenopsylla cheopis* **in Lincoln, Nebraska.** [Research Notes.] *J. Parasitology.* 1944, June, v. 30, No. 3, 202.

BACILLARY DYSENTERY.

SCADDING, J. G. **Bacillary Dysentery.** [Correspondence.] *Lancet.* 1944, Sept. 9, 357.

Referring to BOYD'S criticism of Scadding's paper [this *Bulletin*, 1944, v. 41, 755], the latter reaffirms his opinion that the cases of dysentery treated by him were clinically mild, giving reasons for that view. He also points out that his paper dealt with the *relative* efficacy of the three sulphonamides tested and that no conclusion regarding the efficacy of sulphonamide treatment generally in bacillary dysentery was possible since there was no untreated control group of cases. With regard to the cause of the reduction in the severity of bacillary dysentery in the Middle East since 1940, the author does not agree with Boyd's view that it was due entirely to the early administration of sulphonamides, but thinks that there has been a progressive decline in severity in each succeeding summer. If, however, the diminished severity was really due to the drugs, then the severe cases seen in 1940 and 1941 would have had a favourable course if large doses of ordinary sulphanilamide had been given to the patients ; the author finds this difficult to believe. *J. F. Corson.*

AMOEBIASIS AND INTESTINAL PROTOZOAL INFECTIONS.

DIAZ-RIVERA, R. S. & RASBERRY, E. A. **Amebiasis : Analytical Study of the Cases admitted to a Philadelphia Hospital during the Last 5 Decades.** *Amer. J. Med. Sci.* 1944, June, v. 207, No. 6, 754–9.

Amongst 61,574 admissions during the last five decades to the medical services of the Hospital of the University of Pennsylvania, 32 cases were diagnosed as amoebic dysentery, 4 of which were complicated by hepatic abscess ; but, of 4,764 deaths during this period, only one was regarded as due to amoebic dysentery. At the same time, out of 122,933 surgical admissions with 5,233 deaths, one only was due to amoebic hepatic abscess. Thus only 0·05 per cent. of all medical admissions were cases of amoebiasis, though it has been shown that the carrier rate amongst students, food-handlers and employees of institutions varied from 5 to 10 per cent. In seven cases only did it appear that the disease had been contracted in Philadelphia or nearby, and the conclusion is reached that many cases were probably missed because not enough stools were examined, as, out of the 32 cases, five were

diagnosed and treated during the last six months. It would appear that intensified interest in the disease has been responsible for increase in the numbers recently diagnosed. Of nine cases admitted after 1933, two acquired the infection during the Chicago World Fair and one in Poland.

It is considered that an increase of amoebiasis is to be expected in the United States after the war as the disease will be brought into the country by service men returning from the tropics. *P. Manson-Bahr.*

YATER, W. M. **Diagnosis of Liver Abscess by means of Thorotrast Hepatosplenography.** *J. Amer. Med. Ass.* 1944, July 15, v. 125, No. 11, 775–8, 5 figs.

As a result of service abroad in the armed forces by considerable numbers of the population the author expects a great increase in amoebiasis in the United States.

Coincident with this increase, liver abscess is likely to become commoner; its clinical diagnosis is often difficult and the author reports that Thorotrast hepatosplenography may prove of great value. A 10-year follow-up study of 286 personal cases examined by this method revealed no evidence of any harmful effect.

The author's procedure is slowly to inject 75 cc. of Thorotrast intravenously; X-ray exposures of the upper abdomen being made some hours later. To simplify the injection the Thorotrast is diluted with several hundred cc. of isotonic sodium chloride solution, and given by the gravity method. If peri-venous injection of Thorotrast is made, unsightly hard nodules form around the vein, which later become painful and require excision. These nodules do not become malignant.

The normal liver and spleen appear as homogenous shadows of about the same density as the vertebrae. Space-occupying lesions appear as clear areas in this shadow. Metastatic carcinomas appear as areas of lessened opacity, with clear-cut margins and often with a halo of condensed liver tissue. The author reports five cases of probable amoebic abscess and two cases of pyogenic abscess of the liver among 20 examined. In all these cases hepatosplenography had proved of great value in diagnosis.

[Apart from the difficulty of obtaining Thorotrast in Britain hepatosplenography has not achieved great popularity here because the substance has been shown experimentally to be carcinogenic in animals. Yater's follow-up of cases examined by this method should help to dispel the fear of this danger.] *Eric Samuel.*

RELAPSING FEVER AND OTHER SPIROCHAETOSES.

ORDMAN, D. **Public Health Aspects of an Outbreak of Tick Relapsing Fever in Non-Europeans in Kimberley.** *South African Med. J.* 1944, Aug. 12, v. 18, No. 15, 259–61, 2 figs.

The description of an outbreak of relapsing fever in African and Eurafrican troops at Kimberley.

During the three months ending February 1944, there were 18 laboratory-confirmed cases and 11 cases diagnosed clinically. A careful study of the history of these cases supports the view that the

disease was acquired locally. None of the barracks visited showed any signs of tick infestation, and it seems probable that the infection must have been acquired in civilian dwellings in Kimberley, and *Ornithodorus moubata* full of blood and infected with relapsing fever spirochaetes were collected at one of the " Location " houses visited. Local civilian cases of the disease in non-Europeans undoubtedly occur and may have been overlooked in the past. The author gives details of two cases at Kimberley Hospital, in both of which the blood was negative for spirochaetes by microscopical examination, but spirochaetal infection was demonstrated by biological tests.

In order to control the infection in Kimberley, it is recommended that new " Location " houses should be built of a type that will not readily harbour ticks and that existing houses should be destroyed as soon as possible, preferably by means of fire. *E. Hindle.*

SAPRE, S. N. **Observations on the Biology of** *Ornithodorus papillipes* **Birula.** *Indian J. Vet. Sci. & Animal Husbandry.* 1943, June, v. 13, Pt. 2, 162–5.

RANDALL, R. & COOPER, H. K. **The Golden Hamster** (*Cricetus auratus*) **as a Test Animal for the Diagnosis of Leptospirosis.** *Science.* 1944, Aug. 11, 133–4.

The authors find that young golden hamsters, three to four weeks old are very susceptible to infection with *Leptospira canicola*, whilst young guineapigs and mice are comparatively resistant.

This organism was first isolated in young hamsters after the injection of centrifuged urine from an infected dog whose serum agglutinated *L. canicola* in a dilution of 1 : 2,000. The sediment was injected intraperitoneally into four young hamsters and four young guineapigs. Within nine to ten days the hamsters died of leptospirosis whilst the guineapigs remained normal in appearance. On its second passage in hamsters marked icterus appeared in three to four days, and the animals died five to six days after inoculation. This strain has been labelled " Strain A " and was used by LARSON in his paper on Leptospirosis in hamsters [see *Bulletin of Hygiene*, 1944, v. 19, 768].

Subsequently the owner of the dog from which this strain had been isolated became seriously ill with *canicola* fever, the diagnosis being based on serological tests and finding the leptospira in the urine.

A second strain (" B ") of *L. canicola* was also isolated from a dog by the use of young hamsters and the owner also became infected and developed an agglutination titre for *L. canicola* of 1 : 1,000.

In another case *L. icterohaemorrhagiae* was isolated from an infected dog by the inoculation of venous blood, or the deposit from centrifuged urine, into young hamsters. This is the first instance of the isolation of *L. icterohaemorrhagiae* from a dog in the U.S.A.

These results support the view that the golden hamster is the animal of choice for the isolation of Leptospirae, especially of *L. canicola*, as it produces a fatal infection in young hamsters. It can also be used for the differential diagnosis of *L. canicola* and *L. icterohaemorrhagiae* since the hamster succumbs to both infections, whilst in young guineapigs only the latter produces a fatal infection. *E. Hindle.*

HEILMAN, F. R. & HERRELL, W. E., with the technical assistance of Constance CARTER & Nellie GREENBURG. **Penicillin in the Treatment of Experimental Leptospirosis Icterohaemorrhagica (Weil's Disease).** *Proc. Staff Meetings Mayo Clinic.* 1944, Feb. 23, v. 19, No. 4, 89–99, 2 figs. [15 refs.]

A record of the successful treatment of Weil's disease in guineapigs by means of penicillin.

Young guineapigs weighing 200 gm. were used and infected with a virulent strain of *Leptospira icterohaemorrhagiae*, which caused death in from 6 to 10 days after inoculation. Both calcium and sodium penicillin were used for treatment, and it was found that young guineapigs receiving from 1,000 to 5,000 units of penicillin daily in divided doses often died after several days. The susceptibility of individual animals to the toxicity of penicillin varied greatly and also varied with the dose.

Preliminary experiments were made with 14 treated guineapigs and 14 controls, using 3,000 to 5,000 units of penicillin daily for the treated animals. All the 14 controls showed typical infections, whilst of the 14 treated animals none showed any signs of leptospirosis. Subsequently the dose was reduced and 800 units daily of calcium penicillin adopted in a final experiment involving 64 guineapigs, 32 treated and 32 controls. The treated animals received 800 units of calcium penicillin each day, 200 units at 9.00 a.m. and at 3.00 p.m., and 400 at 9.00 p.m., the drug being suspended in sesame oil. None of the treated animals died of leptospirosis, but three died from effects of penicillin. All the controls became infected and 29 of them died of the disease.

These results suggest that penicillin may be of use in the treatment of Weil's disease and other leptospiral infections in man. *E. Hindle.*

HEILMAN, F. R. & HERRELL, W. E., with the technical assistance of Constance CARTER & Nellie GREENBURG. **Penicillin in the Treatment of Experimental Infections with *Spirillum minus* and *Streptobacillus moniliformis* (Rat-Bite Fever).** *Proc. Staff Meetings Mayo Clinic.* 1944, May 17, v. 19, No. 10, 257–64.

The authors inoculated 50 mice with a strain of *Spirillum minus* isolated from a seven-year-old child suffering from rat-bite fever. Ten days after inoculation 25 of these mice were treated with sodium penicillin at the rate of 1,000 units per day in divided doses, treatment being continued for seven days. All these treated mice became negative for spirilla the morning after treatment began. In a control series of 25 mice all showed spirilla throughout the whole experiment, which lasted 37 days, except one mouse which was negative on the last day.

These results confirm previous observations as to the susceptibility of *Spirillum minus* infections to treatment with penicillin. [See this *Bulletin*, 1944, v. 41, 293.]

In three experiments a total of 86 mice were infected with *Streptobacillus moniliformis*, one strain isolated from a case of rat-bite fever in a 47-year-old man, and two strains isolated from the infected middle ear of rats infected with "turning sickness"; of 43 untreated mice 42 died, 33 within the first two days, whilst out of 43 mice treated with penicillin in divided doses of 1,000 units per day, none died. In the case of two experiments, involving 35 mice, treatment was continued

for seven days and none of the mice developed any signs of arthritis ; but in a third experiment with 8 mice treated for only 5½ days, four developed swelling of one or more joints.

These studies suggest that penicillin may also be of use in the treatment of human cases of rat-bite fever due to infection with *Streptobacillus moniliformis.* *E. Hindle.*

MUDROW, Lilly & BOCK, Marianne. Der Antagonismus von Sulfonamiden und Para-Aminobenzoesäure bei experimentellen Spirochäten-, Spirillen- und Virus-infektionen. [**The Antagonism of Sulphonamides and *p*-Aminobenzoic Acid in Experimental Infections by Spirochaetes, Spirilla and Viruses.**] *Ztschr. f. Immunitätsf. u. Exper. Therap.* 1943, Dec. 30, v. 104, No. 6, 463–73.

Sulphapyridine exerted only a weak therapeutic action upon a laboratory strain of *Sp. duttoni* in mice ; a strain of *Sp. usbekistanica* (used for similar experiments by FELDT [see this *Bulletin*, 1942, v. 39, 344]) was more sensitive. Sulphapyridine given on the second and third days after infection in doses of 1/150 gm. per 20 gm. mouse subcutaneously, or 1/75 gm. orally, cut short the period in which spirochaetes were present in the blood. Sulphathiazole was less effective. If *p*-aminobenzoic acid was given at the same time as the sulphapyridine, the therapeutic action of the latter was almost or completely suppressed, showing that *p*-aminobenzoic acid is antagonistic to the antispirochaetal action of sulphapyridine. [This demonstration of antagonism by *p*-aminobenzoic acid is in conflict with the experience of FELDT (*loc. cit.*) and of the reviewer (this *Bulletin*, 1944, v. 41, 854.)]

The effect of sulphapyridine was also tested on mice infected with *Spirillum minus.* If a single dose of sulphapyridine, 1/200 gm. per 20 gm. mouse, was given by mouth on the second day after inoculation, the infection was completely cured ; the curative effect of 1/300 gm. was less complete. [NITTI *et al.* (this *Bulletin*, 1943, v. 40, 57) have also shown that infections of *S. minus* in the guineapig can be cured by sulphanilamide.] If *p*-aminobenzoic acid was given at the same time as the sulphapyridine, the curative action of the latter was abolished. The infection could also be cured by a single dose of sulphadiazine 1/1,000 gm. per 20 gm. mouse, and this curative action was similarly abolished by *p*-aminobenzoic acid.

In a third series of experiments mice were infected intracerebrally with the virus of lymphogranuloma inguinale ; most of the mice died in 5–10 days. The mice were protected against this infection by sulphapyridine, 1/200 gm. per 20 gm. mouse, by mouth, or by sulphadiazine, 1/600 to 1/25,000 gm. subcutaneously. The therapeutic action was abolished by administering *p*-aminobenzoic acid at the same time.

Mice were also infected by intranasal instillation with the virus of mouse bronchopneumonia ; they died after 4–7 days with characteristic changes in the lungs. Upon this infection sulphapyridine (1/200 gm. subcutaneously) exerted a slight therapeutic action, and sulphadiazine (1/6,000 gm. subcutaneously) conferred complete protection. Their therapeutic action was incompletely abolished by the simultaneous administration of *p*-aminobenzoic acid. *F. Hawking.*

LEPROSY.

HABER, H. **Two Cases of Leprosy in London.** *Lancet.* 1944, May 13, 629–30.

Both patients were Cypriots and both had suffered from syphilis. The first, a male aged 27, came to England in 1935, and while under treatment for syphilis was noticed to have small nodules on his face and subsequently on his chest. Microscopical examination revealed numerous Hansen's bacilli in a nodule, and they were also found in his nasal mucus. Both ulnar nerves were thickened at the elbow. The second patient was also a Cypriot who arrived in this country in 1939 and gave a history of syphilis twelve years before. The case was one of mixed type with patches of anaesthesia and weakness of the hands. The face showed numerous reddish-brown nodules in which were found acid-fast bacilli; they were present also in his nasal discharge. The histamine test was positive in both cases. The disease in these two patients is in an infective stage and they are living in humble circumstances in an overcrowded district. As leprosy is not notifiable they cannot legally be isolated. The author thinks that revision of the law on this point is overdue. *L. Rogers.*

CRAWFORD, W. **An Introduction to the Study of the Incidence of Leprosy in West China.** *Leprosy in India.* 1944, Jan., v. 16, No. 1, 3–5.

Little definite information is available regarding the incidence of leprosy in West China with the exception of the report of a short trip by Dr. MAXWELL in 1935 [see this *Bulletin*, 1937, v. 34, 362], in collaboration with whom the present writer sent maps to missionaries in these parts with the request that they would enter on them the known cases of the disease; the results are now reported for an area bounded by 102 to 105 degrees east longitude and 27 to 38 degrees north latitude. It includes many races but the evidence available shows almost equal incidence in different tribes. Altitude seems to have little effect as cases were met in both high altitudes and in low-lying plains. Nor did humidity or diet appear to play any great part in this area, so climatic conditions have little influence. He agrees with Maxwell that leprosy is a rural disease as most of the cases met with in towns had originated in the country, so, in order to eradicate the disease, it must be attacked in the leper villages. The quality, but not the kind, of houses has some influence. *L. Rogers.*

RAO, A. S. **Report on Leprosy Surveys in Hyderabad, Deccan.** *Leprosy in India.* 1944, Jan., v. 16, No. 1, 24–8, 1 map.

In 1929 and 1940 SANTRA made surveys in some villages of the Nizam of Hyderabad's territories and found leprosy rates of from 0·8 per cent. to 1·6 per cent. In 1943 the present surveys were sanctioned over a period of two years and the findings in five districts are reported in this paper. House-to-house visits were made in villages within five miles of a leprosy hospital or clinic for cases and the contacts of any found were also examined. Propaganda was carried on at the same time to stress the importance of early treatment and the isolation of the infective cases. Tables are given of the number and

types of the cases found in each district together with the age incidence. The incidence varied between 0·11 per cent. and 0·87 per cent. ; 75 per cent. were in males, but the excess is partly due to the prevalence of the purdah system limiting the number of females examined. The proportion of lepromatous cases varied between 16 per cent. and 37 per cent. in different areas ; it was found that the proportion of lepromatous cases was highest in the area in which the total incidence was lowest, but the number examined was low in this district so this may be a coincidence. The proportion of cases in children under 15 was rather low ; namely from 10 per cent. to 14·3 per cent. The author concludes that the leprosy problem is not a serious one in the areas surveyed, but the Editor points out the proportion of lepromatous cases is fairly high in most of the districts surveyed. *L. Rogers.*

SANTRA, I. **Epidemiological Leprosy Surveys in Bihar.** *Leprosy in India.* 1944, Jan., v. 16, No. 1, 29–35.

Bihar is a rather humid province with a high leprosy rate, so the intensive surveys now reported are of interest. Three areas, including one mainly inhabited by Santals, among a few of whom a high rate had previously been found, were dealt with. The incidence in the three areas varied between 2·06 and 3·94 per cent. and among the Santals in two areas it was 2·18 and 2·5 per cent. respectively, which does not show any excess. In persons up to 14 years of age the rates varied between 7·93 and 17·83 ; at 15 to 34 years of age between 36·94 and 46·03 and at above 34 years of age the percentage varied from 45·22 to 51·45. Lepromatous cases varied between 10·67 and 19 per cent. and the highest proportion of lepromatous cases and of those in children was met with in the area with the highest total incidence. Males formed from 52 per cent. to 55 per cent. of the total. In one area an earlier survey by District Board workers had underestimated the incidence. Of cases found in the 1940 survey only 5 of 20 doubtful cases had become definite neural cases. Of 93 neural cases 2 had become lepromatous, 9 had cleared up and 6 had died. Of 30 lepromatous ones seen again 2 had become tuberculoid, 1 now showed no signs and 6 had died, and the remaining 21 were still lepromatous.

L. Rogers.

LEPROSY IN INDIA. 1944, Jan., v. 16, No. 1, 41–6. **Annual Report of the Madras Provincial Branch of the B.E.L.R.A., 1942-43.**

The work in Madras remained under Dr. R. G. COCHRANE and dealt largely with leprosy in childhood, of which he has made a special study at the Saidapet Clinic. During the year about 5,000 more people have been surveyed, with the result of confirming the former finding that contact with leprosy cases is the most important single factor leading to the acquirement of the disease. In a recent analysis of 300 children, open case contact in 69·7 per cent. was traced to an intrafamilial or house contact. In 167 cases there was room-contact, 32·3 per cent. developed the more benign form ; in 141 of these 167 children satisfactory histories were obtained and it was found that 70 open cases had infected them. Hence, of the total of 225 children exposed by close contact, 62·7 per cent. (141) contracted leprosy and some of the remainder may still develop signs of the disease.

Clinical investigations showed that the earliest lesions of leprosy are macules. In prevention night segregation has been continued, but its value is difficult to estimate, although the fewest new cases have been found in the villages where it has been most used, but their population is small.

Treatment of early lepromatous cases is shown to have been successful by the discharge of 141 cases in the year as compared with 123 in the previous year. A post-graduate course was attended by 14 doctors.

L. Rogers.

SANTRA, I. **A Report on an Epidemiological Leprosy Survey in Bombay Presidency.** *Leprosy in India.* 1944, Apr., v. 16, No. 2, 80–85.

In continuation of previous intensive leprosy surveys in small selected areas in various parts of India, to obtain more accurate ideas of the incidence and epidemiology of the disease, the author now reports an inquiry in East Khandesh, Bombay Presidency, where rough surveys of eight districts by the provincial leprosy officer had shown the disease to have the high incidence of 0·5 per cent. The village of Hingona had 3,580 inhabitants, 51 of whom, 1·42 per cent. were found to be infected ; 19·7 per cent. of the cases were of the more serious lepromatous type. The frequency of the disease increased with age and only 16 per cent. were in children below 15 years of age, all being of the neural type ; an unusual feature was a higher incidence in male than in female children ; in adults there was the usual higher rate in males of 60 per cent. of the cases. It is of interest to note that any person suspected of having a patch of leprosy is advised to bathe in the local Tapti river on religious grounds. There is also a local custom of isolating cases of leprosy in their homes in a separate room or outside the village, whatever the type of the disease, and 7 out of the 51 detected cases, 3 of them infectious ones, had been so isolated outside their homes, and 10 of the 17 previously known cases had been living in separate rooms in their own houses. It may prove practicable to isolate all the infective cases in view of this custom. *L. Rogers.*

SANTRA, I. **Epidemiological Leprosy Surveys in the Central Provinces.** *Leprosy in India.* 1944, Jan., v. 16, No. 1, 36–40.

In this survey the data in two different areas are contrasted. The Kurud area is inhabited by depressed or aboriginal rice-eating Hindus in East Central Provinces. In the westernly Kashikhed the people are better-to-do Juar[millet]-eating and Maharatti-speaking with a better diet. The main data are shown in the following table :—

	Kurud	Kashikhed
	Per cent.	Per cent.
Gross incidence	1·16	4·8
Percentage of lepromatous cases	20	8
Percentage of males	59	64
Percentage of cases in children up to 14 ...	14	24

Among the Kurud people the proportion of lepromatous cases is higher, the incidence in children lower and the total incidence lower. These data do not support the commonly held view that a high proportion of lepromatous cases indicates that leprosy is on the increase, but

rather indicate that a high proportion of cases in children suggests that the disease is on the increase, as held by others. The author considers that the disease in the Kurud area is of old standing, but not very serious ; whereas in the Kashikhed one leprosy is more common and probably on the increase ; therefore there is more need for anti-leprosy work in the westernly Berar division than the easternly Chattisgarh one. *L. Rogers.*

DE SOUZA-ARAUJO, H. C. O combate à lepra no Brasil. Balanço de 40 anos de atividades. [**Forty Years of Leprosy Work in Brazil.**] *Brasil-Medico.* 1944, Apr. 22 & 29, v. 58, Nos. 17/18, 158–75.

MARIANO, J. Lepra conjugal (estudo epidemiologico). [**Marital Leprosy; Epidemiological Study.**] *Brasil-Medico.* 1944, Apr. 1, 8 and 15, v. 58, Nos. 14–15–16, 105–9.

Among the many lepers in the Santa Fé Colony are 33 married couples. Seventeen of these were leprous on admission, the remaining 16 form the subject of this article. The history is given of each of these ; the date when the first sign was noticed in husband or wife, and how long afterwards signs appeared in the other. The percentage of conjugal lepers in relation to the total interned at the colony is 2·75 which is close to the figures recorded by others (2·5–5·0. MONTOYA & FLORES ; 1–5 ROGERS & MUIR), but there are exceptions, such as Hawaiians 8·8, Filipinos 1–8. All sixteen of these couples came from rural districts ; all the women were engaged in domestic work ; of the men 13 were agricultural workers ; 27 of the 32 were white, 5 were coloured. As regards ages : between 20 and 30 years there were four men and seven women ; between 30 and 40 years seven men, four women ; between 40 and 50 years three and four respectively, and between 50 and 60 years two and one. The length of cohabitation ranged between three and 34 years. *H. Harold Scott.*

WALLACE, C. A. **Leprosy Infection in Children.** *East African Med. J.* 1944, Mar., v. 21, No. 3, 73–5.

The author, after previous experience of leprosy in India, is in charge of a leper home in Tanganyika to which a number of children are admitted. He stresses the importance of preventing the infection of children with leprosy if the incidence of the disease is to be controlled. Children vary in their degree of immunity to the disease, those in which it is strongest either remaining well or contracting the mild neural form, those with no immunity developing the lepromatous type. Leprous children are delicate and any disease they develop decreases their resistance to the infection. Puberty is the most dangerous age when the infection is liable to progress and become permanent. Over-treatment with hydnocarpus oil, with the production of a severe reaction, is dangerous, but when the treatment is controlled by noting weight and temperature and maintaining nutrition it is beneficial. Any macules of doubtful nature in the child of leprous parents should be watched very carefully and occasional small doses of hydnocarpus oil injected into the lesions. *L. Rogers.*

TROUT, C. L. **The Cultivation of the Lepra Bacillus.** *J. Trop. Med. & Hyg.* 1944, Feb.–Mar., v. 47, No. 1, 1–2.

Ibarra Perez, R. & Gonzalez Prendes, M. A. Orquitis, epididimitis y otros factores que disminuyen la fecundidad en los sujetos leprosos. [**Orchitis, Epididymitis and other Factors producing Sterility in Lepers.**] *Rev. Leprologia, Dermatologia y Sifilografia.* Marianao, Cuba. 1944, July, v. 1, No. 3, 112–21, 1 chart. [38 refs.]

Viamonte Cuervo, J. M., Alonso Suarez, J. E., Castro Palomino, J., Grau Triana, J. & Romero Jordan, O. Sobre las alteraciones oseas en la lepra. [**Bone Changes in Leprosy.**] *Rev. Leprologia, Dermatologia y Sifilografia.* Marianao, Cuba. 1944, July, v. 1, No. 3, 148–54, 3 figs.

Salazar, C. S. Lepra tuberculoide. [**Tuberculoid Leprosy.**] *Rev. Méd.-Quirurg. de Oriente.* 1944, Mar., v. 5, No. 1, 3–7.

Report of a case.

Dharmendra & Santra, I. **The Use of Iodised Hydnocarpus Oil in the Treatment of Leprosy.** *Leprosy in India.* 1944, Apr., v. 16, No. 2, 54–7.

It has long been customary to add creosote as an antiseptic to hydnocarpus oil and its esters for use in the injection treatment of leprosy. Owing to the wartime difficulty in obtaining creosote a search has been made for a substitute. The addition of 1 per cent. thymol to the oil has been found satisfactory, and the following study of the addition of iodine has also been made in view of the long use of iodized esters in the Philippines, where it was found to reduce considerably the irritation caused by injection of the esters. A modification of the method of adding iodine to the oil which was used by Cole [this *Bulletin*, 1930, v. 27, 335] in the Philippines has been found satisfactory. A weighed amount of pure iodine is reduced to a fine powder with a glass pestle and mortar, a small amount of the oil added and grinding continued and the mixture transferred to a flask with the addition of more oil. The corked flask, with a thermometer through the cork, is put on an oil bath and heated gradually to 120°C. and more slowly to 140°C. and maintained for 30 minutes at this temperature. When cool, the iodized oil is filtered, tested to exclude the presence of free iodine and then sterilized in ampoules in an autoclave. The mixture is not more viscid than plain hydnocarpus oil and is suitable for injection. It has been used by the subcutaneous, intramuscular and intradermal methods twice a week in doses of from 2 to 5 or even 10 cc. up to 10 doses, with but little pain and a moderate degree of induration on intradermal injection, and no pain after intramuscular or subcutaneous injection. No abscess or ulceration has followed its use and only slight hyperpigmentation after its intradermal use. A rise of temperature and other reactions have been carefully watched for, but nothing beyond a slight rise of temperature and itching has been noted. The authors conclude that the iodized oil is quite suitable for injection in leprosy and the iodine appears to have some antiseptic action. *L. Rogers.*

Roig, J. T., Acuña, J. & Rodriguez de la Cruz, J. M. Las flacurciáceas Cubanas. Investigaciones acerca de la posibilidad de utilizar los aceites extraidos de las especies cubanas en el tratamiento de la lepra. [**The Flacurciaceae of Cuba. The Possible Use of Extracted Oils in the Treatment of Leprosy.**] *Rev. Leprologia, Dermatologia y Sifilografia.* Marianao, Cuba. 1944, July, v. 1, No. 3, 137–42.

ALEXANDER, V. P. **Treatment of the Neural Symptoms in Leprosy.** *Leprosy in India.* 1944, Jan., v. 16, No. 1, 10–11.

This brief note advocates the following treatment in neural leprosy : (1) 1–2 oz. of wheatgerm flour (" bemax ") and 1–3 oz. of ground nuts taken by mouth daily ; (2) the injection of 1 to 2 cc. of sterile 25 per cent. solution of magnesium sulphate into a nerve sheath, or 2 to 5 cc. or more around the nerve, for pains ; and (3) 60 grains of sodium bicarbonate dissolved in 50 cc. of sterilized saline intravenously in the treatment of neuritis due to lepra reaction. *L. Rogers.*

BOSE, D. N. **Treatment of Leprosy complicated by Syphilis.** *Leprosy in India.* 1944, Jan., v. 16, No. 1, 6–9.

This paper is based on trials in the Asansol Mining Settlement, where syphilis is very common and from 16 per cent. to 20 per cent. of leprosy cases under treatment give positive Kahn reactions, with or without any history or clinical evidence of syphilis. Owing to the poverty of the people arsenical preparations were too expensive and Avenyl (Burroughs, Wellcome & Co.), a mercury preparation dissolved in hydnocarpus oil, gave discouraging results; so the following bismuth preparations were used. Bismuth salicylate or bismuth oxysalicylate (Howard's) were used as 3 per cent. suspensions of the powder in 4 per cent. creosoted hydnocarpus oil or its esters, made up by mixing in a sterile pestle and mortar, putting into a clean stoppered phial and sterilizing in an oil bath kept at 130°C. for half an hour. This preparation was injected intramuscularly in an initial dose of ½ cc., increased weekly by ½ cc. to a maximum of 4 cc. up to a maximum total course of about 4 gm. A table of twelve cases indicates that in nine of them a positive Kahn test was converted into a negative one, and in the other three, 3+ and 4+ reactions were reduced to 1+ or 2+. It is noteworthy that none of the twelve cases showed any clinical signs of syphilis and only four gave a history of the disease. *L. Rogers.*

FAGET, G. H. & ROSS, Hilary. **Evaluation of Positive Kolmer and Kahn Tests in Leprosy.** *Venereal Dis. Information.* 1944, May, v. 25, No. 5, 133–7.

This paper is an important contribution on the much disputed question of the significance of positive serological reactions in leprosy. While most leprologists agree that positive reactions occur in leprosy in the absence of syphilitic infection, a few still regard such reactions as an indication of additional syphilitic infection requiring active treatment before the leprotic infection is likely to yield to treatment, and they maintain that the number of positive reactions in leprosy patients is not greater than the average proportion of syphilitic infections in the general population of the country in question. The careful observations now reported from the U.S.A. National Leprosarium at Carville are summarized in Table I (below).

Both tests gave remarkably similar results and 47·3 per cent. of 676 cases were positive to both tests. The percentages positive in the different types of leprosy were respectively : tuberculoid 10·7, neural 17·6, lepromatous 60·6, and mixed 55·8. It is further shown that among 239 patients submitted to repeated tests, in 147 the results remained practically unchanged upon each re-examination ; as a rule these

TABLE I.

Positive Kolmer and Kahn tests among 695 *leprous patients following their admission to the National Leprosarium.*

Type	Kolmer test			Kahn test			Combined Kolmer and Kahn tests		
	Number examined	Positive		Number examined	Positive		Number examined	Positive in one or both tests	
		No.	Per cent.		No.	Per cent.		No.	Per cent.
Tuberculoid	27	2	7·4	27	2	7·4	28	3	10·7
Neural	141	18	12·8	146	20	13·7	148	26	17·6
Lepromatous	200	110	55·0	196	109	55·6	203	123	60·6
Mixed	308	147	47·7	310	150	48·4	317	177	55·8
Total	676	277	41·0	679	281	41·4	696	329	47·3

patients had shown no marked changes in the activity of the disease during the periods of observation. On the other hand in the remaining 92 patients who were repeatedly tested definite increases or decreases in the degree of reaction were met with ; in 68 of these with a tendency to become more positive. With few exceptions the increase in the serological reaction was accompanied by a definite increase in the clinical manifestations of the disease and *vice versa*. Moreover, when the clinical improvement continued to the point of arrest of the disease, the Kolmer and Kahn tests both reverted to negative. Thus the serological reactions during the course of leprosy are closely correlated with changes in the activity and clinical manifestations of the disease. This suggests the presence of an antibody in the serum of leprosy patients capable of reacting positively with Kolmer and Kahn antigens in the absence of syphilis. Nor did evidence of primary or secondary syphilis develop in any of the patients to account for the change in the serological reactions. In twelve cases a negative reaction gave place to a positive one coincidently with the advance of the leprotic disease to the mixed stage. In another twelve cases in which a positive reaction became negative, in ten of them the disease became arrested at about the same time. In sixteen cases with positive reactions antisyphilitic treatment failed to alter the reaction. They also found increased globulin in the blood of leprosy patients to an even greater extent than in syphilis to account for the positive reactions. They therefore do not regard positive reactions in leprosy as an indication for antisyphilitic treatment in the absence of symptoms of the latter disease. *L. Rogers.*

FIELDING, J. W. & COCHRANE, R. G. **A Plea for the Standardization of the Lepromin Test.** *Med. J. Australia.* 1944, Apr. 8, v. 1, No. 15, 313–15. [16 refs.]

The authors summarize recent work on the lepromin test and deal fully with its standardization by means of counting the lepra bacilli in the preparations used. They support the present view that the reaction is one of bacillary protein alone and is not related to tissue cells. FIELDING (*Australian. J. Exper. Biol. & Med. Sci.*, 1934, v. 12, 1) found that alkalinized lepromin is more effective. The present authors confirm the finding of DHARMENDRA [this *Bulletin*, 1942, v. 39, 855] that lepromins obtained by grinding the bacilli produce much earlier reactions. Lepromins prepared from rat leprosy nodules contain many more bacilli than do those made from human leprosy nodules. The authors describe the Breed counting method for milk and suggest a combination of the Breed method with the Owen dust-counting method to estimate the number of lepra bacilli per cc. in lepromins in order to standardize the material. The original paper should be consulted by those interested in these methods, which are rather complicated. A table is given of the counts obtained by different workers for both human and rat lepromins, using different methods ; these show very variable results. Hence the necessity of using standard methods such as those described in the paper. These have been carefully checked until they yielded fairly reliable results and enable more uniform and reliable reactions to be obtained by the use of this valuable preparation. *L. Rogers.*

FIELDING, J. W. **The Lepromin Test in Laboratory Animals.** *Med. J. Australia.* 1944, May 13, v. 1, No. 20, 439–41. [12 refs.]

The author reports on lepromin tests in rabbits, guineapigs, dogs, cats and rats. He used human and rat leprotic tissues, which are mixed cellular-bacillary suspensions, and also extracts of the acid-fast bacilli prepared by the method of DHARMENDRA [this *Bulletin*, 1942, v. 39, 855], and he confirmed the findings of that worker that bacillary extracts produce earlier reactions than do emulsions of leprotic tissues. In the case of rats all the reactions were negative and in the remaining animals used the results were variable. Emulsions of fresh and of alkaline-fixed tissues showed only slight differences. Two tables of results are given but are not easy to follow, but his general conclusion is that there appears to be some evidence or resistance in different animals, although this is uncertain and it is not always revealed by the lepromin test. *L. Rogers.*

MOM, A. M. & BASOMBRIO, G. Estudio comparativo entre la leprominorreacción y la intradermorreacción por el 2–4 dinitroclorobenceno, en enfermos de lepra, convivientes y controles sanos. (Segunda comunicación.) [**The Lepromin and Dinitrochlorobenzene Reactions compared in Lepers, in those living with them, and in Healthy Controls.**] *Rev. Argentina Dermatosifilologia.* 1944, June, v. 28, No. 2, 165–8, 2 figs.

For the lepromin reaction the authors used standard lepromin prepared according to Muir's method and the bacillary prepared by the Férnandez-Olmos Castro technique; the 2–4 dinitrochlorobenzene was used in injections of 0·1 cc. of a 1 : 1,000 solution in acetone. The result was noted in 48 hours and after three weeks, and, if necessary, after 4, 5 and 6 weeks. An erythema of 1 cm. diameter was designated *one plus*, of 2 cm. or over *two plus*, for the early reaction; for the late reaction a nodule of 5–10 mm. diameter was *one plus*, of over 10 mm., or softening and central ulceration of the nodule, *two plus*.

Among 147 persons thus tested, the three reactions agreed in 91·8 per cent. of the early reactions and 96·4 per cent. of the delayed. Of the different groups of cases :—

1. In 61 *lepromatous* patients, four reactions were doubtful, the rest negative and the agreement between the lepromin and the dinitrochlorobenzene was absolute. In this group were five "burnt-out" cases, in two of which the early reaction was doubtful, the delayed *one plus*.

2. *Tuberculoid* cases, 38 in number. Concordant results were given in 32 of the early reactions and 37 of the late; six of the former gave lepromin positive, dinitrochlorobenzene negative or doubtful.

3. Nine patients, not characterized; both early and late reactions were in harmony in eight, not agreeing in one (lepromin positive, dinitrochlorobenzene doubtful).

4. Of thirteen *cohabiting* with lepers, the reactions, both early and late, agreed in twelve, disagreed in one only (early and late).

5. Twenty-six *healthy controls* were tested for the early reaction and in 22 the results were in agreement; of 24 observed for results of the delayed reaction 22 were in accordance; discordant results (lepromin positive, dinitrochlorobenzene negative) were seen in four early and two late reactions.

In the discussion which followed this paper Professor BALIÑA remarks that, apart from the intrinsic interest of the facts reported,

there are two other problems calling for study in this connexion, namely explanation of the mechanism of the Mitsuda [lepromin] reaction and also to what exactly is due the positive reaction in one case and the negative in another. *H. Harold Scott.*

Basombrio, G. & Mom, A. M. Triple reacción cutanea por el 2–4 dinitroclorobenceno. [**Triple Skin-Reaction to 2–4 Dinitrochlorobenzene.**] *Rev. Argentina Dermatosifilologia.* 1944, June, v. 28, No. 2, 162–4, 1 fig.

During an investigation of certain skin reactions the authors observed a curious phenomenon. Two men, healthy controls, received, intradermally in the abdomen, injections, one of them injections of lepromin and of a 1 : 1,000 acetone solution of 2–4 dinitrochlorobenzene, the other of the latter only. In 48 hours a reaction like the early tuberculin reaction appeared at the injection sites ; on the fourth day there was a localized erythema at the site of the withdrawal (some distance from the actual injection) where a drop of the dinitrochlorobenzene had come into contact with the skin. In another 24 hours these last were vesicular. A week later in various places on the abdomen and the forearms there were erythemato-squamous spots, of the size of a lentil to that of a copper two-centavo piece, becoming larger and resembling a papular syphilide and by the tenth day a nodule (like that of the delayed Mitsuda reaction) with a tuberculoid structure, not clearing up until three weeks had elapsed. *H. Harold Scott.*

Dharmendra. **Intradermal Tests with Antigens prepared from the Urine of Cases of Leprosy.** *Leprosy in India.* 1944, Apr., v. 16, No. 2, 58–61.

In 1940, Berny and Mauzé [this *Bulletin*, 1941, v. 38, 24] described an intradermal reaction in leprosy cases following the injection of an antigen prepared from the urines of bacteriologically positive cases of the disease ; they claimed 100 per cent. positive results in cases of all types of leprosy and negative ones in all controls. The antigen was extracted from the urine, free from albumin, by adding three times the volume of 95 per cent. alcohol. The present paper records an attempt to verify their conclusions, but with negative results. The substance extracted from urines of positive leprosy cases was found to be of the nature of secondary proteoses giving a positive Biuret reaction, but whether extracted from the urines of leprous or of healthy persons, they produced no reactions on intradermal injection in either lepromatous or neural cases of leprosy. Similar substances were extracted from the urines of reacting lepromatous cases of the disease ; the extract from one patient produced reactions in both neural and lepromatous, but that from another reacting case produced positive results in only a few of the neural cases tested and in none of the lepromatous ones. The results of Berny and Mauzé therefore were not confirmed. *L. Rogers.*

Burnet, E. Sur les essais d'inoculation de la lèpre humaine aux rongeurs d'après quelques expériences récentes. [**Inoculation of Rodents with Human Leprosy.**] *Arch. Inst. Pasteur de Tunis.* 1942, June, v. 31, No. 1–2, 20–26. [17 refs.]

This is an interesting review of recent attempts to inoculate hamsters and other rodents with human leprosy bacilli and points out the

difficulties and fallacies of such experiments. These are essentially due to human leprosy tissues not infrequently containing other acid-fast bacilli than that of leprosy, such as the tubercle bacillus or various saprophytic organisms. The author has previously recorded an example of the presence of the tubercle bacillus in a hamster inoculated with leprous material, and similar difficulties have occurred in experiments by DE SOUZA ARAUJO [this *Bulletin*, 1941, v. 38, 218], CHAUSSINAND [*ibid.*, p. 699], DUBOIS and GAVRILOF [*ibid.*, 1942, v. 39, 621] and others which are quoted. It is now evident that hamsters (*Cricetus cricetus*) are not easily infected with the human leprosy bacillus. He concludes that human lepromata may contain all sorts of acid-fast bacilli in addition to that of Hansen, which may grow on culture media suitable for that of the tubercle bacillus or produce on inoculation into rodents nodules or abscesses containing them or even lesions disseminated in the internal organs very similar to those produced by the tubercle bacillus itself and which cannot certainly be distinguished histologically from those of human leprosy lesions. For want of exclusion of these fallacies the experimental production of human leprosy in animals has not yet been proved to take place. Even experimental infection by Hansen's bacillus is still very doubtful. *L. Rogers.*

BURNET, E. & CABASSO, V. Recherche d'extraits huileux des bacilles acido-résistants (sur le bacille de Stefansky). [**Researches on Extracts in Oil of Stéfansky's Bacillus.**] *Arch. Inst. Pasteur de Tunis.* 1942, June, v. 31, No. 1–2, 27–30.

The authors report experiments in which the effects of injecting oil extracts of Stéfansky's rat leprosy bacillus into animals have been investigated. The bacilli were extracted over a period of seventy days in paraffin, chaulmoogra and olive oils, then the mixtures were filtered through Chamberland L3 bougies at 50°C., and the extracts injected into the testes of guineapigs and rats, which were killed and examined 60 to 64 days later. The dose of paraffin or olive oil extract was 0·75 cc. for guineapigs and 0·60 cc. for rats ; 0·15 cc. chaulmoogra oil extract was injected into a young rat. No bacilli were found in any of the lesions produced. The lesions noted were : reduction in the size of the inoculated testicle, and hyaline granulations on the surface of the liver and a few also on the spleen, peritoneum or pericardium ; they were very slightly adherent to these organs. They showed microscopically no cellular structure but only amorphous material with fatty substance of an acid-fast nature, in circular lacunae. To the naked eye there were small grey granulations of a pseudo-membranous nature.
L. Rogers.

BURNET, E. & CABASSO, V. Action des extraits huileux filtrés de bacilles acido-résistants (huile de paraffine et B. de Stéphansky). [**Action of Oil Extracts of Stéfansky's Bacillus.**] *Arch. Inst. Pasteur de Tunis.* 1942, Dec., v. 31, No. 3–4, 194–6.

The authors considered that the oily extracts of rat leprosy bacilli used in the experiments recorded in the above paper might not have been completely freed from the bacilli. They therefore repeated them with the following modifications. Only paraffin oil was used, in quantities of four to five times the volume of Stéfansky's bacilli freed as far as possible from tissue. The mixture was kept at 37°C. for only

14 days and then passed through a Chamberland L2 filter candle at a temperature of 50°C. [In another place the L3 candle is referred to in this connexion.] Doses of 0·4 cc. to 0·75 cc. were injected into the testes or into the peritoneal cavities of guineapigs and rats as before, and the animals sacrificed after 6 to 63 days. The oily solutions were stronger than in the earlier experiments and they produced similar lesions on the surface of the peritoneum, the liver and spleen, and occasionally on the lungs, but they were more extensive in the form of loosely attached membranes. Microscopically the lesions contained a fine network of material staining acid-fast as in the former experiment; this was not decolourized even by the prolonged action of 33 per cent. nitric acid and of 95 per cent. alcohol. The effects were similar to those produced by the injection of dead bacilli in oil. No bacilli were found in the lesions. *L. Rogers.*

Tomé Bona, J. M. La lepra. [**Leprosy.**] **Páginas de divulgación para el médico práctico. Primera edición. Opusculos sobre dermosifiliografia. 111 pp., 10 figs. on 2 pls. 1942. Madrid: Ediciones Morata.**

A general account.

HELMINTHIASIS.

Valdés Dapena, A. & Pérez Hurtado, F. Sindrome febril con eosinofilia: Fasciolosis hepática. Reporte de un caso. [**Fever and Eosinophilia associated with Infection with** *Fasciola hepatica.*] *Avance Med.* Habana. 1944, June, v. 5, No. 6, 83–4.

A case of human infestation with *Fasciola hepatica* is recorded; the patient was a white woman, aged 36, a native of Cuba. She had daily fever with rigors and a temperature rising to 41°C. [105·8°F.] and pain and tenderness in the right hypochondrium. There was no nausea, vomiting or diarrhoea. No parasites were found in the faeces. The blood showed eosinophilia of 74 per cent. This led to an examination of the bile, obtained by duodenal tube, and it was found to contain numerous eggs of *F. hepatica.* The results of treatment will be published later. [See also this *Bulletin*, 1944, v. 41, 677; 1943, v. 40, 253 & 472; 1942, v. 39, 14 & 703.] *J. F. Corson.*

Kourí, P. & Angulo, J. J. Un nuevo hospedero de la forma larvaria del *Multiceps serialis.* [**A New Host of the Larval Form of** *M. serialis.*] *Rev. Med. Trop. y Parasit.* Habana. 1944, May–June, v. 10, No. 3, 64–9, 9 figs. [13 refs.] English summary.

"Some characteristics of a *Coenurus serialis*, larval stage of *Multiceps serialis*, found in an autochthonous cuban rodent: the "hutia conga" (*Capromys pilorides* Say), are described. This animal is reported as another host for the mentioned larval stage and this is the first time that this parasite is reported in Cuba."

Davis, H. **Ancylostomiasis associated with Hematuria.** *War Medicine.* Chicago. 1944, June, v. 5, No. 6, 385–8.

A young white American army recruit was admitted to hospital on 26th July, 1943, because of pain in the lower part of his back with, at

times, sudden pain in the back of the right thigh and hip. The pain began suddenly in February 1943, lasted for about two weeks, returned in April, and persisted since then in varying degree ; it finally led to his discharge from the Army. A provisional diagnosis of ruptured intervertebral disc could not be confirmed, but he was found to have symptomless haematuria. Remarkably thorough and complete clinical and laboratory examinations were made and are given in detail in the paper. Eosinophile leucocytes in the blood always formed less than 1 per cent. of the total white cells ; the erythrocytes were over 4 millions per cmm. in July and August, decreased to 3,810,000 by September 24th, and rose again to reach 5½ millions on January 14th. The urine showed erythrocytes, usually about 20 to 50 " per high power field " from the end of August to November ; there were a few pus cells but only a trace of albumin was found. Ova of *Necator americanus* were first seen in the faeces on October 30th.

Treatment.—" The patient was given courses of therapy with hexylresorcinol and tetrachloroethylene on November 6, November 18 and December 6, but hookworm ova continued to appear in the stools. On December 15, 4 cc. of tetrachloroethylene was given through a duodenal tube. This likewise failed to eliminate the ova. On January 5, 1944, 3 cc. of carbon tetrachloride mixed with 15 Gm. of magnesium sulfate was given. Results of all subsequent examinations of urine and stools were negative."

The author concludes that the urinary abnormalities were caused by the hookworm infestation although a nephrotic syndrome was absent.

J. F. Corson.

ARANGO, E. G. Anemia por ancylostomiasis. [**Hookworm Anaemia.**] *Rev. Facultad de Med.* Bogota. 1944, Feb., v. 12, No. 8, 389–96.

When one gramme of faeces contains 10,000 hookworm eggs, by Stoll's method of examination, anaemia sets in and, if there is no other cause for the anaemia, it is of the hypochromic, microcytic type, and reticulocytes are present in greater numbers than in other types of anaemia. It is not, moreover, due to haemolysis, according to the authors, but to continued loss of blood from the ulcerous or other mucosal lesions of the intestine. The degree of eosinophilia present bears no direct relation to the severity of infestation. The authors mention, in a list of cases, one with 4,000 ova per gramme of faeces with 10 per cent. eosinophiles, while another with 51,000 ova had only 5 per cent. Regeneration of the blood is slow, particularly in the earlier stages when the red cell count is between one and three million per cmm. The immediate cause of death in cases of severe infestation and anaemia is cardiac dilatation, and the fatal issue is often heralded by a drop in the reticulocytes and in the eosinophile percentage ; *vice versa*, when treatment is going well the eosinophiles show an increase. When anthelmintics are administered, such as oil of chenopodium or thymol, the total red cell count falls, but not if, before and after their administration, liver extract is given. [It will be remembered that other workers have stressed the importance of malnutrition in the causation of anaemia in hookworm infestation, see NAPIER *et al.*, this *Bulletin*, 1942, v. 39, 100 ; HILL & ANDREWS, *ibid.*, 1943, v. 40, 324.]

H. Harold Scott.

NAPIER, L. E. **Filariasis due to** *Wuchereria bancrofti*. *Medicine.* 1944, May, v. 23, No. 2, 149–79, 6 figs. [18 refs.]

This is a general account of filariasis due to infection with *W. bancrofti*, in a form which would be suitable to a text-book on tropical medicine. It is not a record of new work, but a systematic description, in which well established facts are restated, along with original observations from the author's own experience, and references to the recent findings of other workers. The author is a master of his subject, and the writing is clear and lucid. Nothing of importance has been omitted, but the details of surgical technique in the removal of elephantoid tissue does not come within his scope. The article is apposite at the present time, when so many white troops are engaged in countries in which filariasis is endemic, and readers will obtain a good clinical picture, with adequate description of the pathology and treatment of the various clinical conditions, from this account. *Charles Wilcocks.*

WRIGHT, W. H. & MURDOCK, J. R. **Intradermal Reactions following the Use of** *Dirofilaria immitis* **Antigen in Persons Infected with** *Onchocerca volvulus*. *Amer. J. Trop. Med.* 1944, May, v. 24, No. 3, 199–202. [10 refs.]

The authors required to know, for the purposes of research on and control of onchocerciasis in Guatemala and Mexico, the number of people who might be carriers of the infestation and yet showed no evidence of external nodules. Intradermal tests were employed and the antigen used was prepared by BOZICEVICH, of the National Institute of Health, by extraction of *Dirofilaria immitis* in physiological saline; it contained 0·5 per cent. phenol as a preservative. Control tests were done with physiological saline and a canine serum to rule out positive reactions possibly due to sensitivity to any protein in the antigen derived from the host of *Dirofilaria*. Before the intradermal injections, scratch tests were done on all patients to avoid the possibility of intense local reactions and anaphylactic shock. These eliminated two patients. The patients had had onchocerciasis for periods varying from 1 to 20 years and were either of pure Indian or Mestizo (half-breed) blood. Of the 20 tested, 19 showed microfilariae in biopsies of the skin. There were no demonstrable nodules in 17, but these had had external cysts removed a few months before the tests. Three had cysts at the time of the tests. No information was available about their Wassermann or Kahn reactions. Probably some were syphilitic and "nearly all" had malaria. All had infestations with *Trichuris*, hookworms, *Strongyloides stercoralis*, *Ascaris* or combinations of these.

All of the 20 subjects reacted positively to antigen dilutions of 1 : 2,000. Ten out of eleven reacted to dilutions of 1 : 4,000. The negative one reacted well to 1 : 2,000 and had been infested for 20 years and was blinded by a microfilarial invasion of the eye. Two were positive to the canine serum control (one to 1 : 2,000, the other to 1 : 4,000), but the reactions of both were much stronger to the *Dirofilaria* antigen.

Of 20 control patients harbouring *Trichuris*, *Enterobius*, hookworm, *Ascaris*, *Wuchereria bancrofti*, *Entamoeba histolytica* or *Dientamoeba fragilis* or combinations of these, 11 were positive to a 1 : 2,000 dilution of *Dirofilaria* antigen, while 7 out of 19, tested with a dilution of 1 : 4,000, were positive. Four of these controls were tested at Huixtla;

of these one had always lived in an endemic onchocerciasis zone, one had visited endemic zones more than once, both being positive to a 1 : 2,000 dilution. The other two were the authors and both were negative to the *Dirofilaria* antigen, although both were positive to a 1 : 2,000 dilution of the canine serum. The 16 other controls were tested in Washington and had never been exposed to infestation with *Onchocerca*, although all had intestinal helminths. Of these, 9 were positive to a 1: 2,000 dilution and 7 to 1 : 4,000. Of all the controls other than the authors, three were positive to the canine serum at 1 : 2,000 and two of these were positive to *Dirofilaria* antigen at 1 : 2,000. There was no correlation between the species of helminth in the controls and their reaction to the antigen. The authors conclude that care is needed in the evaluation of intradermal tests. Further tests with more dilute solutions of the antigen are needed to determine whether it is any use for diagnosis. The evidence indicates that there would not be enough protein material derived from the dog host of *Dirofilaria* to cause false positives. None of the patients had a history of allergy, so that allergic states were not involved ; and Bozicevich and Hutter (below) have shown that most allergic individuals do not react to this *Dirofilaria* antigen in dilutions above 1 : 4,000.

G. Lapage.

Bozicevich, J. & Hutter, A. M. **Intradermal and Serological Tests with *Dirofilaria immitis* Antigen in Cases of Human Filariasis.** *Amer. J. Trop. Med.* 1944, May, v. 24, No. 3, 203–8.

The authors tested subjects suspected of infestation with *Wuchereria bancrofti*. After reviewing the literature and describing in detail their method of making the *Dirofilaria* antigen used, the authors describe the results of the injection of 0·01 cc., an amount sufficient to raise the smallest possible weal. A reaction was considered positive when the diameter of the antigen weal exceeded that of the control weal by 3 mm. The reactions obtained were immediate, appearing within a few minutes and reaching their maximum in 15 minutes, or delayed, the latter occurring only occasionally and always in subjects who had shown an immediate reaction. A dilution of the antigen at 1 : 1,000 gave false positives in about 30 per cent. of individuals not exposed to *W. bancrofti* and the same concentration of the protein control antigens used (canine serum : *Trichinella* antigen) also produced reactions in some individuals. But at 1 : 2,000 the antigen was more specific and this dilution screened out most positive reactions in non-infested subjects. A number of allergic individuals were included in the controls in order to exclude false positives due to protein carried over from the host of the worm in individuals sensitive to such protein. In these allergic subjects the 1 : 2,000 dilution produced a number of false positive reactions, but a dilution of 1 : 4,000 eliminated most of these. To test the influence of pyrexia on the reactions tests were done with a dilution of 1 : 8,000 on six patients with malaria who had not been exposed to *W. bancrofti*. One of these was positive to both *Dirofilaria* antigen and to dog serum and was classed as negative ; one was positive to dog serum but not to *Dirofilaria* ; one was positive to *Trichinella* but not to the other antigens. Tests done on 25 subjects who had been exposed to infestation with *W. bancrofti* and who showed clinical symptoms of this, although microfilariae could not be found, showed that all were positive to 1 : 8,000 dilutions of *Dirofilaria*

antigen. But 6 were positive to dog protein in the same dilution and two were positive to *Trichinella* antigen, while one reacted to all the antigens and to the saline control as well.

For passive transfer studies blood was taken from three cases of "filariasis" who gave positive reactions to a 1 : 8,000 dilution of *Dirofilaria* antigen, but no reactions to the control substances. Six recipients of this serum, five of whom had never been outside the continental limits of the United States, each received four injections of 0·1 cc. of the serum intradermally, and 24 hours later these sites were respectively injected with 0·01 cc. of *Dirofilaria* antigen, dog protein, *Trichinella* antigen (all at 1 : 8,000) and with physiological saline. Controls were done with these materials on the left arm which had not had any serum. In the prepared sites the *Dirofilaria* antigen gave positive results in four out of the six subjects. On the control left arm there was no reaction to *Dirofilaria* or *Trichinella* antigen or to the saline, but one subject, who had handled laboratory dogs for over a year, was positive to dog protein.

Complement fixation tests with antigen in dilutions of from 1 : 100 to 1 : 1,600 and serum diluted up to 1 : 16 or undiluted were negative, although positive results were obtained with rabbits immunized with the residue from the antigen preparation. An alcoholic *Dirofilaria* antigen appeared to be inferior to the saline one. Seven out of twelve rabbits thus immunized died, and this seems to indicate that the adult worms contain some toxic substance. This substance may be connected with the fact that the intradermal tests were followed by an exacerbation of symptoms of lymphangitis in seven subjects and by pain in the scrotum and lymph glands, especially the inguinal glands, in eight subjects. The authors conclude that the *Dirofilaria* antigen at a dilution of 1 : 8,000, if it is used with the control materials mentioned, is useful for the "diagnosis of filariasis," but they do not agree that the dilutions of 1 : 200 used by TALIAFERRO and HOFFMAN [this *Bulletin*, 1931, v. 28, 214] or 1 : 200 and 1 : 1,000 used by FAIRLEY [*ibid.*, 1931, v. 28, 679; 1932, v. 29, 427] are reliable for diagnosis. The authors emphasize the necessity of a dilution of *Dirofilaria* antigen which will not give positive reactions due to infestations with nematodes other than *W. bancrofti*. *G. Lapage.*

LAMY, L. Intensité et vitesse relatives de la formation des dispositifs capteurs chez les Hyphomycètes prédateurs de Nématodes. [**Intensity and Speed of Development of Hyphomycetes Predatory on Nematodes.**] *C. R. Soc. Biol.* 1943, June, v. 137, No. 11–12, 337–9.

See this *Bulletin*, 1939, v. 36, 833.

DESCHIENS, R. & LAMY, L. Conditions pratique de culture, de sporulation et de récolte des spores d'Hyphomycètes prédateurs de Nématodes. [**The Cultivation of Hyphomycetes Predatory on Nematodes.**] *C. R. Soc. Biol.* 1943, June, v. 137, No. 11–12, 381–3.

DEFICIENCY DISEASES.

JAFFÉ, R., in collaboration with K. NEUBÜRGER. Beriberi como causa de muerte en Venezuela. [**Beriberi as a Cause of Death in Venezuela.**] *Rev. Policlinica Caracas.* 1944, Jan.–Feb., v. 13, No. 74, 7–14.

[Not a very satisfactory paper, in that the evidence adduced is—the authors themselves acknowledge it—insufficient for a decision to be reached.] The authors state that in some 10 per cent. of cases showing evidence of myocarditis at autopsy there is no proof of the presence of syphilis, schistosomiasis or ankylostomiasis, and that these *may* be due to beriberi. In other words, the myocarditis is insufficient to warrant a diagnosis of beriberi. If, at the same time, there are signs of neuritis the diagnosis is more likely, but still not absolutely certain. They give notes of examination of the heart and of the nerves of the legs of 13 " suspicious " cases, but in four the nerves showed, microscopically, no change. In the others there were indications of syphilis, or of schistosomiasis or hookworm infestation, and, in one patient, pyloric carcinoma, so that no case of uncomplicated beriberi came to histological examination. Consequently, the authors conclude that it is highly probable that in Venezuela there are cases of beriberi with anatomical lesions so grave as to cause death, for some show cardiac dilatation without myocarditis but " with changes corresponding to those described in medical literature in cases of avitaminosis " ; that cases of myocardial infiltration occur for which no obvious cause can be assigned [hardly a satisfactory reason] ; that myocardial lesions may be associated with signs of degeneration in the peripheral nerves of the legs. *H. Harold Scott.*

DINIZ, O. Observações e ligeiras notas acerca de cinco casos de pelagra. [**Notes on Five Cases of Pellagra.**] *Brasil-Medico.* 1944, Feb. 19 & 26, v. 58, Nos. 8 & 9, 51–8, 8 figs.

It was generally held in the principal medical centres of Brazil that the incidence of pellagra was very low. The same was true of the rural areas as the result of an enquiry in 1933. Since then the disease has been found to be not so rare in certain provinces as was formerly supposed, possibly owing to faulty diagnosis in the past and deterioration of nutrition more recently. The author gives notes upon five ordinary cases of pellagra which will serve to interest practitioners in Brazil. *H. S. Stannus.*

GILLMAN, T., GILLMAN, J., INGLIS, J., FRIEDLANDER, L. & HAMMAR, E. **Substitution of Whole Stomach Extract for Vitamins in the Treatment of Malignant Infantile Pellagra.** [Correspondence.] *Nature.* 1944, Aug. 12, 210.

In three years almost 300 children suffering from acute malnutrition have been admitted to the non-European hospital in Johannesburg ; more than 60 per cent. showed clinical signs of pellagra. The syndrome included oedema ; pellagrous skin lesions ; bulky, pale stools containing much unsplit fat ; extremely low serum albumin and globulin ; mild microcytic anaemia ; diffusely fatty liver. The authors have devised an improved liver biopsy technique, and have found that the microscopic appearance of the liver is the best guide in assessing the severity of the condition.

This condition has been described in Uganda by TROWELL [this *Bulletin*, 1941, v. 38, 722], in the Gold Coast by WILLIAMS [*ibid.*, 1934, v. 31, 344], in South Africa by MACVICAR and by KARK [*ibid.*, 1944, v. 41, 418], and the present authors have found, as Trowell did, that vitamin therapy failed to avert death in a large proportion of cases. They record the results of treatment in 20 children, 7 of whom were treated with thiamin, nicotinic acid or brewers' yeast (with 6 deaths), 7 with crude liver extract injected twice daily for 7 days (with 2 deaths), and 6 with 10 gm. ventriculin (Parke Davis) plus 10 cc. of N/10 hydrochloric acid daily, in one dose, for 5 days (no deaths). The response to crude liver was slow, but that to ventriculin was dramatic, although these children were as severely affected as the others; the liver was almost free of fat within two weeks.

In 7 cases of severe pellagra in adults, ventriculin was found to be a much more rapid and effective treatment than nicotinic acid or other vitamins, though the mental symptoms responded slowly.

[Much has recently been written on malnutrition in Africa, and there seems to be no doubt about the seriousness of the problem.]

Charles Wilcocks.

GILLMAN, T. & GILLMAN J. **Mitochondrial Origin of Cytosiderin (Iron Pigment) in the Liver of Human Pellagrins.** [Correspondence.] *Nature.* 1944, July 29, 148.

The terms haemosiderosis and haemochromatosis have in the past focussed attention on the metabolism of haemoglobin as the cause of the accumulation of iron in the body tissues.

Using an improved liver biopsy method the authors have studied the liver at the time of admission and during therapy of 76 non-European pellagrins—21 children under seven years of age and 55 adolescents and adults. The presence of masses of iron-containing pigment in liver cells at one stage or another they regard as a constant feature in pellagra among adults but it is not found in children under nine. The iron pigment, they believe, arises within the cells of the liver, first in the region between the nucleus and the biliary pole of the cell, corresponding to the position occupied by the Golgi apparatus. They therefore speak of cytosiderin.

By means of certain techniques the authors demonstrated simultaneously iron-containing pigment and Sharlach-staining fat in one section and iron, fat and mitochondria in another. From this it is argued that the granules of cytosiderin developed from mitochondria, which passed through a lipo-protein stage to form combined and free iron. Cytosiderin is carried to the portal tracts and, when there is extensive hepatic cytosiderosis, the lymph glands in the porta hepatis assume a deep brown colour. With the progress of this disease the portal tracts become thickened, leading ultimately to typical pigment cirrhosis. The fatty liver of pellagra may go on to pigment cirrhosis.

H. S. Stannus.

BEAN, W. B., SPIES, T. D. & VILTER R. W. **A Note on Irradiation Sickness.** *Amer. J. Med. Sci.* 1944, July, v. 208, No. 1, 46–54, 6 figs. (2 coloured).

In 1938 it had been demonstrated by the authors that nicotinic acid was of use in combating the symptoms of irradiation (X-rays)

sickness—nausea, vomiting, headaches, cramps and diarrhoea. The behaviour of urinary pigments and the codehydrogenases I and II after irradiation of the spleen resembled that in severely ill pellagrins.

In their more recent investigation the effects of a standard dose of irradiation was studied in (*a*) normal well-fed subjects, (*b*) those given a diet deficient in vitamin B, with or without supplements of vitamin, (*c*) pellagrins.

"Persons on a diet poor in the vitamin B complex developed roentgen sickness, which could be prevented or reduced in severity, by giving supplements of nicotinic acid or thiamine for a few days before irradiation."

Once the reaction was established exhibition of these substances was relatively ineffective.

The authors are of opinion that no very definite conclusions are justified from these results. *H. S. Stannus.*

DERMATOLOGY AND FUNGOUS DISEASES.

Symmers, D. & Sporer, A. **Maduromycosis of the Hand, with special reference to heretofore Undescribed Foreign Body Granulomas formed around Disintegrated Chlamydospores.** *Arch. Pathology.* 1944, May, v. 37, No. 5, 309–18, 6 figs.

A white man aged 67 fell and injured his right hand with splinters from the floor boards of a house in New York. Two or three weeks afterwards the hand became greatly swollen, and later developed numerous sinuses, connected with hard deep-seated nodules, from which pus containing black, gunpowder-like, grains was discharged. The lesion was a typical mycetoma of one of the black grain varieties. A short course of intensive treatment with potassium iodide caused an acute inflammatory reaction in the lesions and was not continued. The duration of the disease, at the time of publication, was about seven years, and X-ray examinations made at intervals over a period of six years showed progressive bone lesions consisting of rarefaction of some of the carpal bones and the heads of the metacarpals, with productive periostitis of the shafts of some of the metacarpals and the proximal phalanges. The causative fungus was not identified [the brief account given suggests that it may have been a species of *Madurella*], nor did the authors succeed in cultivating it. A description of the histology of the lesion adds little to our previous knowledge. *J. T. Duncan.*

Alonso, J. M. & Cancela Freijo, J. Histoplasmosis de Darling. Primera observación efectuada en el Uruguay. Curación clínica luego de tratamiento con sulfadiazina. (Nota preliminar.) [**The First Case of Histoplasmosis in Uruguay: Clinical Cure after Treatment with Sulphadiazine.**] *Arch. Uruguayos de Med., Cirug. y Especialidades.* 1944, Mar., v. 24, No. 3, 193–210, 5 figs. English summary.

TROPICAL OPHTHALMOLOGY.

A Review of Recent Articles. XLIV.*

Trachoma.—The difficulties associated with experimental research in trachoma are exemplified by the observation of Bland[1] that grivet and vervet monkeys frequently suffer from a spontaneous folliculosis indistinguishable from the conjunctival reaction induced in them by the inoculation of trachomatous material. These monkeys are, however, susceptible to trachoma, and, though the virus retains its capacity to infect man after four passages through the monkey, inclusion bodies are not to be found in the animal's conjunctiva. The inclusion bodies reappear in the human subject infected from a grivet of the fourth passage concurrently with the development of clinical trachoma.

Burnet[2] and others have reported experiments in which they have infected mice with trachoma virus, by peritoneal and intranasal inoculation, and have produced a condition closely resembling trachoma by inoculating the conjunctiva of human subjects with an emulsion of infected mouse lung tissue. No macroscopic or microscopic changes could be observed in the infected mice, but emulsions of their lungs and spleens caused a reaction in the chorio-allantoic membrane of the chick similar to that caused by the inoculation of the rickettsiae of epidemic typhus. This reaction continues to be produced after repeated alternating passages through mouse and chick.

The fact that the trachoma block of the L.C.C. Hospital at White Oak has been closed owing to lack of patients is evidence of the value of good hygiene and efficient school medical service in preventing the disease. Sorsby,[3] in recording this event, pleads that the disease should be made notifiable and that statutory provision be made for treatment centres. In a review of the efficacy of the treatment of trachoma by the sulphonamides, Rohrschneider[4] remarks that mass treatment by the oral administration of an efficient drug would be a great advantage as it would effect a considerable saving in the services of trained medical men. Differing opinions have been expressed regarding the efficiency of the sulphonamides in the treatment of the disease. Uncertainty of diagnosis, variation of the disease in different geographical localities, and the occasional occurrence of spontaneous cure render it difficult to estimate the value of treatment by the drugs, but the majority of observers report in favour of sulphanilamide therapy. Some consider that, although complete cure does not result, the conjunctiva is freed from secondary infections and the trachoma rendered quiescent, so that recovery is facilitated. Others claim that trachoma can be cured completely by treatment with the drug alone.

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* For the 43rd of this Series see Vol. 41, pp. 509–512.

[1] Bland, J. O. W. Spontaneous Folliculosis of the Conjunctiva in Grivet and Vervet Monkeys (*Lasiopyga griseoviridis* syn. *Cercopithecus aethiops* and *L. pygerythra* syn. *C. pygerythra*) and the Susceptibility of the Grivet to Trachoma Virus. *J. Path. & Bact.* 1944, Apr., v. 56, No. 2, 161–71, 4 figs. (2 coloured) on 2 pls. [16 refs.]

[2] Burnet, E., Cabasso, V., Cuénod, A. & Nataf, R. Inoculations expérimentales du virus trachomateaux. *Arch. Inst. Pasteur de Tunis.* 1942, Dec., v. 31, No. 3–4, 165–78.

[3] Sorsby, A. Trachoma in London : the End of a Chapter. *Brit. Med. J.* 1944, Aug. 12, 220–21.

[4] Rohrschneider, W. Die Sulfonamidbehandlung bei der Bekämpfung des Trachoms. *Deut. med. Woch.* 1943, Nov. 12, v. 69, No. 45/46, 769–71. [10 refs.]

Very prolonged observation, however, is necessary before one can be sure that relapse will not occur. At a special trachoma hospital at Soldau 151 cases of the disease were treated, 126 by sulphanilamide alone and 25 with additional local treatment. Apparent cure was obtained in 83, improvement in 32, and 11 were unaffected. The author's own experience leads him to conclude that the sulphonamides have a specific action in the disease and that the improvement observed is not merely due to the elimination of secondary infections. He has found no essential difference in the efficacy of the various preparations and has had as good results from protracted treatment with small doses continued over a long period as from intensive treatment with large intermittent doses. For a man weighing 70 kgm. he has used albucid [sulphacetamide] in a daily dose of 1·5 gm. for two weeks followed by an intermission of two weeks—total 126 gm., and in a daily dose of 4·5 gm. for one week followed by seven days' intermission—total 126 gm. He has found sulphanilamide therapy particularly effective when combined with the ordinary local treatment of the disease.

Cornea.—AYOUB[5] has described a form of keratitis among the troops of the 8th Army when fighting in the desert during the summer of 1942. Patients complained of photophobia and profuse lachrymation with smarting and itching of one or both eyes. There was moderate ciliary injection, and a band of roughened corneal epithelium could be seen in the area exposed by the palpebral aperture when the eyes were "screwed up." This band was greyish but translucent and stained with fluorescein. The lesion was punctate and might be limited to a few lines of greyish dots. Full mydriasis was found to relieve the discomfort. Recovery occurred in five or seven days. The author considers that the condition is probably due to a dryness of the corneal epithelium induced by a hot, dry wind and suggests that the use of goggles would prevent its occurrence. He remarks, too, that desiccation may be a more potent factor than excessive ultra-violet light reflections in the causation of snow-blindness.

GALTON[6] has reported good results in the treatment of *perforating corneal ulcer* by the local and oral employment of sulphanilamide together with the excision of any prolapsed iris and the use of a conjunctival flap. He has treated fifteen cases; twelve of these had had trachoma and eight suffered from entropion and trichiasis. The lachrymal sac was apparently healthy in every case. Malnutrition was a predisposing factor in the causation of the disease. The eyes were cleaned for three or four days by irrigations with 1–10,000 perchloride of mercury lotion and by insufflations of sulphanilamide powder. After the conjunctival flap had been fashioned the prolapsed iris was freed with a spatula from the edges of the ulcer and then drawn out and excised. Retraction of the membrane ought to occur. The conjunctival sutures usually cut out after five or six days.

H. Kirkpatrick.

PESSÔA, S. B. Profilaxia do tracoma no Estado de S. Paulo. [**Prophylaxis of Trachoma in S. Paulo.**] *Brasil-Medico.* 1944, Apr. 22 & 29, v. 58, Nos. 17/18, 140–47, 1 map & 1 fig.

[The title of this article hardly covers the contents. The author describes the epidemiology of the condition, its prevalence in different

[5] AYOUB, J. E. M. Dessication Keratitis. *Brit. J. Ophthalm.* 1944, July, v. 28, No. 7, 347–55, 5 figs.

[6] GALTON, E. M. G. Treatment of Perforating Corneal Ulcer. *Lancet.* 1944, Aug. 26, 272–3.

districts, by race and age, the cause, the stages of the disease and the measures taken to deal with it from the early years of the present century.]

First, as regards prevalence : optimists estimate the number of cases as 250,000 among a population of seven million, pessimists give more than four times this, "more than a million." The authors strike a happy mean and call it 500,000. The percentage in rural districts is about ten times that of urban, but when the urban figure is high the rural is correspondingly high. The negro seems to be more resistant to infection than does the white man ; predisposing factors are insanitation and overcrowding, and naturally where hygienic conditions are bad children largely suffer. Contagion may be carried by lice or other ectoparasites, by insects such as house flies, *Hippelates* and others, and by direct contact or transmission by soiled towels and the like.

Sulphanilamide is used in treatment with great success. The history of campaigns against the disease is a chequered one. In 1904 a decree was passed ordering the removal and repatriation of all immigrants suffering from trachoma, and in 1906 a Service of Prophylaxis was instituted with Dr. Eusebio de Queiroz at the head, assisted by 56 doctors and 362 laymen. Two years later this was abolished for financial reasons. In 1911 the Service was re-established, with 16 doctors, 32 nurses and two secretaries. In another three years, in 1914, it was again abolished. In 1918 the Governor created four Commissions, consisting altogether of eight doctors, eight nurses and 11 assistants. Six years later this Service was merged with General Health Service and the special work was stopped. Then, in 1938, the Trachoma Service was re-established, but its work was confined to the capital. Finally, in 1941, dispensaries were established and rural posts ; the latter comprised fixed posts and travelling ambulances. In the article a map is given showing where these posts have been set up, but it is on so small a scale that even with a lens the names are not decipherable. In 11 months of 1943, 32 dispensaries and 15 fixed rural posts were established and during that time 47,270 persons were treated, totalling 544,674 attendances, and 984 institutions, schools, crèches, etc., were visited. Of those treated, 41,427 were from the interior of the State and 5,843 in the Capital. *H. Harold Scott.*

MISCELLANEOUS.

LEISHMAN, A. W. D. & KELSALL, A. R. **A Year of Military Medicine in India.** *Lancet.* 1944, Aug. 19, 231–5.

At a large base hospital in Western India 11,645 cases were treated in one year from August 1942. Of these, 51·5 per cent. were of tropical diseases. Most of the patients were British soldiers ; a few were Indian or Anglo-Indian.

Although the hospital was in an area of low endemicity there were 2,819 cases of malaria ; 2,010 of the cases were benign tertian infections, 46 per cent. of which were relapses ; 595 were malignant tertian, including 30 per cent. of relapsed cases ; 12 were quartan, and 51 were mixed infections. The rest were diagnosed on clinical grounds ; most of them had been under treatment before admission.

The too-prevalent practice of starting treatment before finding the parasites is regarded as being "seldom justified." [The word "seldom" deserves emphasis because there are sometimes conditions in which this "unscientific" practice is quite justified.]

Typically-periodic temperatures were exceptional at the onset, so that at this stage the disease was often clinically indistinguishable from the prevalent "undifferentiated short fevers."

Relapses were frequent after the standard Army treatment, which consisted of 30 grains of quinine daily for two days; 0·3 gramme of mepacrine daily for five days; two days rest; and then 0·03 gramme of pamaquin daily for five days. Intravenous treatment was needed in a small number of the malignant tertian cases.

There was one case of acute haemoglobinuria, which occurred on the fourth day of the course of pamaquin; the haemoglobin was reduced to 14 per cent. but there was rapid recovery without transfusion. In another case there was an attack of acute haemolytic anaemia, with a fall of the haemoglobin to 10 per cent.; this occurred early in the treatment, and rapid recovery followed. There were two deaths from cerebral malaria in cases in which repeated intravenous injections of quinine failed.

Persistent anaemia with splenomegaly was rare, even in cases with repeated relapses.

There were 261 proved cases of bacillary dysentery of the following types; the figures in brackets are percentages. Flexner (52·4); Shiga (15·7); Sonne (13·8); Schmitz (8·8); Boyd (8·8); and Newcastle (0·5). In 511 other cases diagnosed as bacillary dysentery the organisms were not isolated, and there were also 509 cases of diarrhoea.

Saline treatment was found to be of definite value; of a small group of 32 cases, half were treated with salines, and the stools became free from blood within two days; the other half, who were not given salines, continued to pass blood for three to four days, and in some of them there was persistent abdominal discomfort.

Supplies of sulphaguanidine were limited, but in most cases recovery was so rapid that little advantage could have been expected from its use. In one case in which succinyl sulphathiazole (94 grammes in five days) failed, there was a prompt response to sulphaguanidine; otherwise little difference was observed in the effects of the two drugs.

There were only three deaths; two in patients with cirrhosis of the liver and one in a patient with chronic refractory anaemia.

Among the 501 cases of amoebic dysentery the diagnosis was confirmed by finding parasites in all but 29. There were 35 cases of amoebic hepatitis and five of amoebic liver abscess. The frequency of amoebic infection was striking; about one-fourth of all the admissions for diarrhoea turned out to be due to this cause. No less than 45 per cent. of the 333 fresh admissions for amoebic dysentery were relapses. Primary attacks yielded promptly to emetine, 6 to 12 grains in all. This was followed by a course of carbarsone, "Amoebiarson," or acetarsol, by mouth for 10 days. In relapses the treatment was less effective, and in cases with several relapses it ultimately failed altogether. Emetine bismuth iodide was not available till near the end of the period, but in 13 cases with persistent relapses a course of this combined with quinoxyl, gave very disappointing results; six of the patients had already relapsed within 21 days [presumably, up to the time of writing]. An urgent need is expressed for a drug that will cure chronic and relapsing cases.

Amoebic hepatitis always responded quickly to emetine; so did four of the cases of liver abscess, in which cure followed a course of the drug and aspiration. In the fatal case there was no response; closed surgical drainage was carried out, and post mortem there was extensive amoebic ulceration of the bowel and the right lobe of the liver was reduced to a mere shell.

In several cases of amoebic dysentery there were granulomata simulating appendix abscess, carcinoma, and other conditions.

In one of the two fatal cases there was perforation of the bowel and intussusception.

A persistent non-specific colitis gradually developed in some of the cases, both of amoebic and bacillary dysentery.

There were 1,134 cases of short fever which ran a mild course of two to seven days; leucopenia was common but the "true picture" of dengue and sandfly fever was not presented. [The term true picture applies only to epidemics of these diseases in susceptible communities; in sporadic attacks in areas of endemicity "typical" pictures may be the exception rather than the rule.]

A chronic fatty diarrhoea occurred in 52 cases; in four of these it followed amoebic, and in seven, bacillary, dysentery. The authors hesitated to call these cases "true sprue" because 25 of the patients had already returned to duty, though in a lower category, apparently cured, and only 18 had been evacuated. [As the usual duration of the treatment was about six months there need have been little hesitation in calling the disease "sprue."]

There were 49 cases of "effects of heat"; 46 of these were heat-exhaustion; the other three patients were admitted for aphasia and signs of cerebral degeneration resulting from heat-hyperpyrexia in which the temperature had risen to 109°–111°F. and unconsciousness had persisted for 4–21 days.

There is a brief reference to 42 cases of tropical typhus with an average duration of 14 days; 23 of the patients were admitted from the local region and 21 of these had a Weil-Felix titre of 1–250 or over. In 13 the titre was highest to *Proteus OX19*; in three to *OX2*; in three to *OXK*, in one to *OX19* and *OX2*; and in one to *OX19* and *OXK*. The reaction never became positive before the 10th day. The Widal reaction was positive in eight cases. There were no deaths. "There was no louse infestation or obvious insect bites, and one is left with the possibility that a flea is concerned."

Within eight months seven cases of tropical eosinophilia were seen. Bronchitis with an increase of the eosinophiles to 41–72 per cent., was the chief feature. Neoarsphenamine was curative. All the patients were Indians or Anglo-Indians from coastal districts.

Infective hepatitis was diagnosed in 992 cases. The average duration of the illness was 29 days. There were no deaths. No cases of the disease without jaundice were recognized.

A very unusual occurrence was the admission of 68 British soldiers suffering from smallpox. There were 10 deaths, eight in unvaccinated patients and two in patients who had been vaccinated in childhood only. Vaccination before the appearance of the rash, carried out as a routine measure, was thought to have aggravated the illness in five of the cases, and one patient who was vaccinated 11 days before the appearance of the rash died of haemorrhagic smallpox. *Staphylococcus aureus* was isolated in pure culture from the pustules of all the patients

who were examined bacteriologically. Sulphathiazole was strikingly effective, but sulphapyridine gave poor results.

There were only two deaths among the 45 cases of enteric-group fever, of which 32 were typhoid and 11 paratyphoid A. In many of the cases proved by culture the Widal reaction was negative, and in one case of smallpox the titre with an O strain of typhoid was 1–640.

Treatment was by insisting on an adequate intake of calories, vitamins, and fluids; this was achieved by untiring efforts on the part of the nursing staff.

There were 59 cases of limb paralysis caused by anterior poliomyelitis; in two acute cases the "iron lung" was used, but both patients died, one of them suddenly a few days after being released from the apparatus.

Among the 1,377 cases of respiratory disease, which included 116 of pneumonia and 56 of pulmonary tuberculosis, there were only two deaths, both from the latter disease. The authors insist that men with a tendency to lung trouble are quite unsuited for service in the tropics, and that the same thing applies to skin diseases, of which there were 1,242 cases.

Among 215 direct admissions for dyspeptic symptoms 23 per cent. were cases of peptic ulcer; 71 per cent. of these ulcer patients had had symptoms before the war, with an average duration of 5½ years, so that there was no evidence that Army life could be blamed for the development of the disease. Other special causes of dyspeptic symptoms were amoebiasis, without dysentery, and worm infestation.

[Altogether this is a record of which the authors have good reason to be proud; the paper ought to be reprinted and circulated to every medical officer serving in India. The standards of treatment and nursing must have been high, seeing that there were only 30 deaths, of which 10 were due to the very exceptional prevalence of smallpox. There were not more than three deaths from any of the great tropical diseases, malaria, bacillary dysentery, and amoebic infection.]

John W. D. Megaw.

APLEY, J. & GRANT, G. H. **Eosinophilia with Pulmonary Disease on Return from the Tropics.** *Lancet.* 1944, Sept. 2, 308–9.

The authors give a very instructive description of the case of an Englishman, aged 37 years, invalided from India on account of a refractory condition known under the names Loeffler's syndrome, eosinophilic lung, and tropical eosinophilia, in which none of the common causes of eosinophilia in the tropics is found, unless it is a form of allergy, a point not yet decided.

The history of this patient is that he had suffered from mild recurrences of winter bronchitis since an attack of pneumonia in childhood. After three months in Bengal [how long ago is not stated, but probably nearly two years] he had a severe "bronchitis" with pain in the chest and troublesome cough; X-rays showed congestion and an opaque area in the left lung and fibrosis in the right. Ten weeks later X-rays showed nothing abnormal, but the cough continued, worse at night. Some six weeks later an eczematous condition set in and, as it proved refractory to treatment, he was sent home to England. Examination of stools, urine and sputum yielded nothing positive to elucidate the cause. During the ensuing two months after his return to England the leucocyte counts ranged from 7,000 to 16,000 per cm.

and the eosinophiles between 6 and 40 per cent., the last when the total was highest. The patient went on leave with his leucocytes 7,000 and eosinophiles 420, or 6 per cent. [Nothing is said as regards his treatment or the subsequent course of his illness.]

The authors draw up the following table, contrasting Loeffler's syndrome and tropical eosinophilia, which would seem to indicate two very distinct conditions, but in the discussion which follows they show that none of these so-called distinctions has any true validity, that they tend to run one into the other and that they are merely merging manifestations of one basal condition [see this *Bulletin*, 1944, v. 41, 693 and 517 ; in the latter several other references are given].

Loeffler's Syndrome	*Tropical Eosinophilia*
1. Recorded in Europeans.	1. In all races.
2. Dry European climate.	2. Tropics, near sea or in humid atmosphere.
3. Mild disease, lasting few days ; temperature raised 1 or 2 days.	3. Acute febrile onset lasting a few weeks ; then becomes chronic.
4. Variable transient eosinophilia (up to 66 per cent.)	4. Variable persistent eosinophilia (up to 89 per cent.).
5. No splenomegaly recorded.	5. Splenomegaly in acute phase.
6. X-ray shadowing of the lungs, variable in type, but always resolving quickly.	6. Disseminate mottling of both lungs in second week of illness ; after acute phase chronic bronchitis.
7. Speedy spontaneous recovery.	7. Chronic, unless treated with arsenic.

H. Harold Scott.

KARK, Emily. **Menarche in South African Bantu Girls.** *South African J. Med. Sci.* 1943, Feb., v. 8, No. 1, 35–40, 1 fig. [12 refs.]

An enquiry was made as to the date of onset of menstruation in 1,038 Bantu school girls in South Africa. The girls came from four different rural areas, two of which are in the Transvaal, one in the Cape Province and one in Natal.

In all the groups investigated it was found that no girl had begun menstruating before 13 and that at 15, 40·5 per cent., at 16, 80 per cent., and at 19, 100 per cent. had commenced.

It was found that menstruation started about one year earlier in the two southern areas than in the two northern, although the climate in the southern areas is colder. This finding, though contrary to popular belief, confirms the work of other investigators, notably that of C. A. MILLS (*Hum. Biol.*, 1937, v. 9, 43.).

Diseases and malnutrition were less common in the southern areas. The exact influence of climate, disease and malnutrition on the age of onset was difficult to assess. The author, however, considers that physique and health are important factors. *M. G. Blacklock.*

VALENZUELA, R. H. El tratamiento del noma. [**The Treatment of Noma.**] *Rev. Mexicana Pediatria.* 1943, Aug. 10, v. 13, No. 8, 310–16. [12 refs.]

During the four years, 1939–1942, 36 cases of gangrenous stomatitis [noma or cancrum oris] were treated in the children's infectious

diseases section of the General Hospital at Mexico ; only six recovered. In 1943 the author treated six cases by the local application of sulphathiazole powder ; it was usually applied three times a day after preliminary cleansing of the area with Dakin's solution. No notes of the cases are given and it is not stated whether all the children recovered, but the author considers that the treatment had a better effect than any other method that he has hitherto used. [See also this *Bulletin*, 1942, v. 39, 411.] *J. F. Corson.*

Gear, J. H. S., Yeo, R. M. & Bodenstein, J. C. **The Aetiology of Onyalai.** *South African Med. J.* 1944, Aug. 12, v. 18, No. 15, 265–6.

This paper is so full of facts that even a long abstract would fail to do it justice. To give a satisfactory account would need extracts rather than an abstract. Onyalai is a bleeding disease characterized by acute onset of haemorrhage from nose, mouth, intestine and urinary tract, purpuric spots in the skin and blood-bullae in the buccal mucosa, the blood showing lengthened bleeding-time, normal coagulation-time, normal prothrombin estimation, but marked thrombocytopenia ; it is known in West and East Africa and in Rhodesia (possibly elsewhere).

The patient is usually a native, but Europeans and others (Chinese, for example) may suffer from the disease, and the authors have found that in some cases at least the patient has recently been taking " native medicine." During the illness this is acknowledged, but on recovery strenuously denied, even though the medicines may be found on the patient, probably from fear of vengeance by the witch-doctor who has supplied them. One patient whose case is recorded had three native medicines, one of which contained fragments of cantharides ; but the pathology of cantharides poisoning is not that of onyalai and the dose of it was found not to exceed the B.P. medicinal dose. It may be, as the authors suggest, that even small doses may, in certain individuals, lead to sensitization manifested as a thrombocytopenia. On the other hand, another of the native medicines was found by animal experiment to contain a haemorrhagic principle. Guineapigs injected subcutaneously in the thigh with 0·25–1·0 cc. of a 1 in 10 emulsion in physiological saline died within 24 hours and extensive haemorrhages were found at autopsy from the injection site spreading even over the abdomen ; also the inguinal glands were enlarged and haemorrhagic, the lungs showed haemorrhagic foci as did also the liver, spleen, the gastric and intestinal mucosa, the adrenals and the kidneys. Further studies are to be undertaken to determine more accurately the nature of the haemorrhagic principle. The results of this work will be awaited with singular interest [see this *Bulletin*, 1938, v. 35, 74, 444 ; 1939, v. 36, 157, 158, 938 ; 1940, v. 37, 675 ; 1944, v. 41, 427].

H. Harold Scott.

Forrest, G. H. **Propamidine. A Report of its Use as an Antiseptic.** *New Zealand Med. J.* 1944, June, v. 43, No. 235, 140–42.

In the Solomon Islands the author treated eight cases of indolent tropical ulcer with 0·1 per cent. propamidine in Mumford base (lanette wax, hard paraffin, technical white oil) or in a water-soluble jelly base,

with excellent results. In some, bed rest and local application of sulphathiazole had been unsuccessful, but propamidine effected apparently permanent cure in 10 days or less. The ulcer was completely filled with the preparation used, covered with a single layer of sterile gauze and the whole covered with a 3-inch strip of elastoplast. Dressings were changed every 24 (later 48) hours.

These applications are useful in the treatment of burns and of early otitis externa. Propamidine possesses many of the advantages of penicillin over the sulphonamides, but requires none of the elaborate precautions to prevent deterioration, a point of some importance in the forward areas. No facilities for bacteriological investigation were available to the author. *Charles Wilcocks.*

Orsini, O. Pequena epidemia familiar de ulcera tropical. [**A Small Familial Epidemic of Tropical Ulcer.**] *Brasil-Medico.* 1944, Mar. 4 & 11, v. 58, Nos. 10 & 11, 72–4, 2 figs. English summary (8 lines).

Six cases occurred in one family in Belo Horizonte. The oldest patient was aged 16, all the ulcers were on the lower leg or foot, and the author contends that the affection was probably spread from one child to another as they lay together in bed, where direct contact was probably frequent. The patient with most ulcers (4) was apparently the healthiest of these children, and it seems, therefore, that poor general health is not a necessary precursor, as has been supposed. Nevertheless, poor condition probably does predispose to ulcer. The ulcers began as pustules; nothing is said of their originating in small abrasions of the skin. *Charles Wilcocks.*

Thiersch, J. B. **Solar Radiation and Pernicious Anaemia in South Australia.** *Med. J. Australia.* 1944, June 24, v. 1, No. 26, 583–4.

Smith (*Amer. J. Med. Sci.*, 1934, v. 188, 200) concluded that a statistically significant relationship existed between the mortality from pernicious anaemia and the relative lack of solar radiation in the United States of America. Askanazy (*Fol. Haematol*, 1937, v. 58, 289) concluded that the incidence of pernicious anaemia in Europe gradually decreased from north to south. Apperly (*Amer. J. Med. Sci.*, 1942, v. 203, 854) also concluded that the mortality rate of pernicious anaemia and solar radiation were inversely related in U.S.A.

Adelaide, South Australia, has a Mediterranean climate with a dry subtropical summer, and the solar radiation there is very much greater than in England. Since the population is mainly of British stock, a comparison can be made between the two countries of the effect of solar radiation on the incidence of pernicious anaemia and cancer of the skin. From a study of the figures bearing on the question the author could find no evidence to support the view of Smith and Apperly regarding pernicious anaemia, but the incidence of cancer of the skin is far greater in South Australia than in the British Isles. As it occurs mainly on the face and hands this suggests that the increase is due to the greater solar radiation in South Australia. *J. F. Corson.*

Fornieles Ulibarri, F. Breve nota sobre un caso de miasis subcutánea producida por la larva de la Hypoderma bovi ? o la Hypoderma lineatum ? [**A Case of Cutaneous Myiasis due to** *H. bovis* **or** *H. lineatum*.] *Rev. Sanidad e Hig. Pública.* Madrid. 1944, Jan.–Feb., v. 18, No. 1, 75–6.

Torres Cañamares, F. Nuevas localidades de "Phlebotomus" en España y algunas observaciones sobre los mismos. [**New Localities of** *Phlebotomus* **in Spain.**] *Rev. Sanidad e Hig. Pública.* Madrid. 1944, Jan.–Feb., v. 18, No. 1, 38–9, 1 map.

Monchadsky, A. S., Blagoveschensky, D. I., Bregetova, N. G. & Ukhova, A. N. [**The Search for New Repellent Anti-Mosquito Substances.**] *Med. Parasit. & Parasitic Dis.* Moscow. 1943, v. 12, No. 5, 56–62. [In Russian.]

Very effective protection from mosquitoes can be secured by nets, impregnated with repellent substances. A series of vegetable oils was examined for suitability for this purpose, and dextra-α-pinene selected as deserving extensive tests. This substance is obtained from *Juniperus seravschanica*, which is widely distributed in central Asia. Oil extracted from the young shoots contains cedrol, which is used for medicinal purposes and, in its purification, d-α-pinene occurs in the waste products. Experiments were made with refined d-α-pinene and (since this is rather expensive) with the following mixtures :—

1. Naphthalysol 20 parts, pinene 10 parts, water 70 parts.
2. Lysol 15 parts, pinene 8 parts, water 77 parts.

Before testing the repellents, a survey of the mosquitoes in the district (Tadjik S.S.R.) was made in August and September 1942. The numbers of the commonest species found were as follows :—

	Open air		In cattle sheds		In dwellings	
	♀	♂	♀	♂	♀	♂
Anopheles superpictus ...	24	0	25	63	191	1
Mansonia richiardii	2,944	0	57	0	52	0
Aëdes caspius	57	0	72	2	16	0
Culex spp.	61	0	47	3	8	0

Attacks of mosquitoes under natural conditions on men wearing nets impregnated with repellents were compared with controls wearing unimpregnated veils. Results :—

Impregnant	No. of tests	No. of mosquito attacks in	
		Tests	Controls
d-α-pinene	14	3	156
Naphtha-lysol-pinene ...	38	8	512
Lysol-pinene	7	1	112

Thus, the repellent power of pinene and the mixtures was well demonstrated.

Further experiments were made in a mud-walled, thatched, native hut to test the effectiveness of the repellent in protecting dwellings. It was found that a coarse net (1·5 cm. mesh) impregnated with pinene and hung across the window very greatly reduced the numbers of mosquitoes found indoors (mainly *A. superpictus*).

J. R. Busvine.

Wagener, H. P. **Toxoplasmic Chorioretinitis.** *Amer. J. Med. Sci.* 1944, Aug., v. 208, No. 2, 255–64. [21 refs.]

Ray, H. N. **A Method of Rapid Staining of Intestinal Flagellates.** *Indian Med. Gaz.* 1944, Apr., v. 79, No. 4, 158, 12 figs. on 1 pl.

The following method, though unsuitable for detailed cytological studies, is said by the author to be "a great aid to the proper understanding of the morphology of the organism."

Place several drops of blood from the finger on a microscope slide and allow them to dry. Take up the material containing the flagellates with a long fine pipette and expel it on to one of the dried drops of blood and allow them to become mixed; then take up the mixture, which should be blood-red in colour, into the same pipette and streak it finely along a clean warm slide so that the streaks dry up immediately. Stain with Leishman or Giemsa stain. Ordinary thin films can also be made with the mixture but they produce a slight distortion of the flagellates. Photomicrographs of flagellates from the intestine of some animals illustrate the results.

J. F. Corson.

Noble, G. A. **A Five-Minute Method for staining Fecal Smears.** *Science.* 1944, July 14, 37–8.

The following method of staining fresh smears of faeces is rapid and very suitable for showing intestinal protozoa, though not so good as standard methods for critical cytological examination. The solutions used are: I, Formalin (10 per cent. by volume) 3 parts; glacial acetic acid 1 part; ferric ammonium sulphate 3 per cent. II, Aqueous solution of haematoxylin, 0·5 per cent. III and IV, Dioxan. V, Dioxan and toluol [toluene], equal parts.

Cover a thin, wet smear of faeces with I and warm until it steams; pour off and add II and warm for a few seconds; wash for one minute in running water in a jar, remove excess of water and pass through III, IV and V, allowing at least one minute's stay in IV. Mount in clarite.

J. F. Corson.

Glazier, M. & Olson, C. **Reno-Ureteral Colic in the South Pacific Area.** *U.S. Nav. Med. Bull.* 1944, July, v. 43, No. 1, 80–82.

Renal or ureteric colic is a common complaint in the U.S. Navy throughout the South Pacific area and, to judge by the diagnoses made, its cause is not well understood.

The authors studied 30 cases admitted to a base hospital during five months of 1943. Haematuria was present in 23, tenderness in the costo-vertebral angle in 16, frequency of micturition, dysuria and other

symptoms of lower urinary tract involvement in 18, and fever in six. In about one-half of the patients *p*H determinations and examinations for crystals were carried out—the *p*H was always low (average 5·8) and the urine was always rich in crystals of uric acid, urates and oxalates. In three cases calculi were found at radiological examination.

The condition is ascribed to excessive sweating and deficient fluid intake. These two factors lead to highly concentrated, highly acid urine, in which the crystals are precipitated, and the haematuria and colic are probably the result of the passage of small calculi or of showers of crystals. [Sulphonamides were not involved in this series, but the danger of their administration in the absence of adequate fluid is evident.] Treatment should consist of rendering the urine alkaline and the administration of large quantities of fluid. [This syndrome was reported in the Royal Navy, much earlier in the war, by Beach (*Bulletin of War Medicine*, 1944, v. 4, 326), whose conclusions are confirmed by the present author.] *Charles Wilcocks.*

Bean, W. B. & Eichna, L. W. **Performance in relation to Environmental Temperature. Reactions of Normal Young Men to Simulated Desert Environment.** *Federation Proc.* Baltimore. 1943, Sept., v. 2, No. 3, 144–58, 16 charts.

This is an important paper, establishing for relatively large numbers of men some of the significant changes which occur during acclimatization to heat.

The subjects were 56 healthy men, mostly between the ages of 20 and 28. They were exposed for one to three weeks in a room at a dry bulb temperature of 120°F. from 8 a.m. to 5 p.m. and then at 90°F. for all but two hours of the rest of the day, the relative humidity in the heat being maintained at 20 per cent. The air movement was moderate, and no additional heat by radiation was involved. In the morning two successive periods of work, and in the afternoon three bouts of work, were performed. The work consisted in one group of marching 2½ miles in 50 minutes carrying a 20 lb. pack, and in the second group of cycling.

The main results are as follows :—

" The unacclimatized man works in the heat with a high pulse rate, a high body temperature and an unstable blood pressure, particularly after change in posture. . . .

" With continued exposure and work in the heat, acclimatization ensues and the heart rate, rectal temperature and blood pressure return to levels approximating those obtained after similar work in cool environments. . . .

" The acclimatized man is alert, performs his work energetically and without symptoms . . . the unacclimatized man working in the heat becomes dull and apathetic, performs his work poorly, has a rapid heart rate and a high rectal temperature and may manifest to varying degrees, and either singly or in combinations, the symptoms and signs of heat exhaustion."

The pulse rate and body temperature were used as indices of acclimatization, and indicated that by the fourth or fifth day in most men a major part of the improvement had occurred. Men in good physical condition generally acclimatize more rapidly. Acclimatization to hot dry environments increases markedly the ability of men to work efficiently in hot moist environments. *J. S. Weiner.*

ADOLPH, E. F. **Physiological Fitness for the Desert.** *Federation Proc. Baltimore.* 1943, Sept., v. 2, No. 3, 158–64, 2 figs. [Numerous refs.]

This paper, a contribution to a symposium on fitness, gives a brief review of the physiology of acclimatization to desert conditions. The well-known fact that some individuals are better than others in withstanding these conditions is discussed. The individual difference is due largely to a greater ability to dissipate heat and depends on such factors as the number and size of active sweat glands. The important changes in acclimatization are, greater ability to carry out work, an increased rate of sweating and a circulatory improvement. The pulse rate is reduced, the peripheral blood flow is maintained with less intravenous pooling and the pulse pressure is reduced. The body temperature increases less during work.

Acclimatization to the desert is rapid. After the first two or three days when the man feels uncomfortable and lazy, a marked subjective improvement occurs. Acclimatization is retained even after several weeks without exposure.

To assess the fitness of a man in desert conditions, performance tests ought to be related to the work the man is actually expected to do. Fitness tests should, in general, include a walking test, a postural test and a manipulative test. The best criteria are probably measurements of changes in rectal temperature, pulse rate, pulse pressure, and sweating. Other factors to be considered are age, resting pulse rate, and the ratio of body surface to body mass. *J. S. Weiner.*

BOOK REVIEWS.

ROGERS, Leonard [K.C.S.I., C.I.E., LL.D., M.D., B.S., F.R.C.P., F.R.C.S., F.R.S., etc.] & MEGAW, John W. D. [K.C.I.E., B.A., M.B., Hon. D.Sc., Queen's University, Belfast, etc.]. **Tropical Medicine.** Fifth Edition. pp. x+518, 2 coloured pls. & 87 text figs. 1944. London: J. & A. Churchill Ltd., 104, Gloucester Place, Portman Square. [21*s.*]

This fifth edition of what is now a classic of tropical medicine has been considerably re-written, and brought thoroughly up to date. The biggest alterations are in the sections on malaria, kala azar, trypanosomiasis, the fevers of the typhus group, leprosy and the dietetic diseases, and the whole is now a very comprehensive and accurate guide to the practice of clinical tropical medicine. Prevention and control of these diseases are not overlooked, but the book is strongest on the clinical and epidemiological sides. As before, the writing and layout are clear and crisp. Much modern work is incorporated, but is set in its place easily and economically; the reader is not confused by over-many small paragraphs recounting the investigations of individual workers, from which to work out his own conclusions—

much of that is done for him. The re-writing has not entailed lengthening the book, though the amount of type on each page is greater than in the previous edition.

Inevitably, perhaps, the strongest chapters are those on diseases to which the two authors have made their own scientific contributions in the past, but these are many. The study of malaria, leishmaniasis, the dengue group, the typhus group, amoebiasis, cholera, leprosy and other diseases has been much furthered by their work, and the evidence of their continuing lively and critical interest is to be found in these pages.

The reviewer is glad to see that amoebic dysentery and amoebic hepatitis are now dealt with in contiguous chapters, instead of being widely separated, as in the previous edition. He would urge that the same linking be made between kala azar and dermal leishmaniasis, which are still dealt with in chapters II and XVIII respectively. The inclusion of sections on diseases due to certain trematodes (other than *Schistosoma*, which is dealt with) of the Far East, would be appreciated by those who in future will work in those areas.

Those new to the tropics should read with care the chapter on climate as a disease factor. The present war has emphasized not only the powerful influence climatic conditions exert on health, but also the avoidable wastage due to insufficient understanding of these effects. Much of this ill-health could be prevented if the facts here so clearly expressed were taken to heart. Doctors going for the first time to the tropics would also be well advised to grasp the implications of the chapter on the incidence of general diseases in the tropics. To Europeans the tropical diseases loom large, but in the indigenous peoples of hot countries, although malaria and dysentery are massive causes of infant death and adult ill-health, the cosmopolitan diseases such as pneumonia, tuberculosis and venereal diseases are more widespread than is commonly realized. These matters are put in good perspective.

Dietetic diseases are clearly discussed; the medical and administrative authorities of tropical countries would do well to note the paragraph on page 407 :—

Dietetic Malnutrition.

" This is so common in many parts of the tropics that it has come to be regarded as the normal condition of the inhabitants. In reality it is the most important of all the tropical diseases; it causes life-long disablement in hundreds of millions of people; indirectly it is responsible for millions of deaths every year. The cause is an insufficient supply of proteins, calories, fats, vitamins, and other substances, needed to maintain growth and health."

When the diet of these unfortunate people is adequate, the treatment and control of tropical diseases will be easier. *Charles Wilcocks.*

TRUBY, Albert E. [Brigadier General, U.S. Army, Retd.]. Memoir of Walter Reed. **The Yellow Fever Episode.** pp. xviii+239, 29 figs. & 1 coloured pl. 1944. London & New York: Medical Book Department of Harper & Brothers. [15*s.*]

Many accounts or sketches of the life of Walter Reed have been written, but none so full, so detailed, so carefully authenticated regarding what may be called the Cuban period, as this by General

Truby. The author first met Walter Reed as a candidate before the Examination Board of which Reed was a member in 1898, and he was in constant, almost daily, touch with him throughout the whole period of the investigation in Cuba. As the subtitle of the book shows, only the Yellow Fever episode of Reed's life is dealt with; no attempt is made to detail his activities prior to the Cuban days, and his post-Cuban days were few. He returned to Pennsylvania in 1901 and for the later years of his life he was far from robust and seems to have suffered from chronic appendicitis; in November 1902 Reed died from peritonitis following operation for this disease and was buried in Arlington Cemetery.

The story has been told so often that we need give only the main outlines of the work of the Board, often, but wrongly, called the Commission. At the time when work was started, in June 1900, typhoid, malaria, dengue and yellow fever were, or had recently been, rife in Cuba. Sanitation was regarded as the concern of the Engineers though the Corps had a medical officer to assist, if called upon, which was not often. Disinfection of rooms was carried out by burning sulphur freely and the next day the rooms were sprayed with perchloride of mercury solution to destroy mosquitoes, while for disinfection of clothing SO_2 and formaldehyde gas were used. On account of malaria every case of fever was isolated and protected from mosquitoes, in accordance with the recent knowledge of the mosquito-transmission of malaria.

Sanarelli's Bacillus icteroides as the cause of yellow fever then held the field; the theory was strongly supported by medical officers of the Marine Hospital Service who had just reported finding the organism in 13 out of 14 cases of the disease in Havana. Reed and Carroll, members of the Board, went thoroughly into the question and showed that Bacillus icteroides was none other than the hog-cholera bacillus with which Reed had previously worked and with which he was quite familiar. They concluded, much to the chagrin of the Marine Service medical officers, that it played no part in the aetiology of yellow fever.

The Board then turned to investigating the mosquito-transmission theory, put forward by Carlos Finlay in 1881, and the question of experimenting on human volunteers came up for discussion. The Governor-General, Leonard Wood, was also a medical man, with progressive ideas, and he gave his assent and approval, provided that the subjects were really volunteers, were of age (24 years old in Cuba) and gave their consent in writing and made no claim on the Government.

New light is thrown on the story of Lazear's unfortunate death in September 1900. The author thinks that the notion, according to the account commonly put forward, of Lazear letting a mosquito bite him in the ward of Las Animas hospital, not recognizing the danger, is not true, but that he deliberately inoculated himself by means of a laboratory-bred infected mosquito and that his notebook indicated this, or the probability of it, but that he kept quiet about it for fear his life-assurance would be forfeited if it became known that he had deliberately infected himself and died of the infection.

Of great interest are the details of the plans for establishing Camp Lazear for subsequent experimental work to test and prove the mosquito transmission theory, and to show that this was the only mode, and not one of several, among them contact with the soiled clothes and bed linen of a yellow fever patient.

In January 1901, Reed reported the results of experimentally injecting the blood of a patient on the first day of his illness into Spanish volunteers and wrote to General Sternberg informing him that "the result was negative" except for "slight headache and general muscular soreness, on the eighth and ninth days," but that the subject had proved resistant to mosquito transmission from another case. Either the slight symptoms were a mild immunizing attack or the man was already immune, or partly so, from living in Havana. Juan Guiteras had already advanced the theory, and was probably the first to do so, that Cuban children were not born immune but acquired immunity from mild, unrecognized attacks in early childhood.

Chapter XII is valuable as an important summary of the parts played by each of the participants in the work carried out, and the credit due to each in the great achievement. This, of course, cannot be done except on broad lines because all worked as a team, but the chief rôle of the constituent members can be stated. Thus, Carlos Finlay was the first to put forward in 1881 the view that yellow fever virus, was conveyed by a mosquito, *Culex fasciatus* (later *Stegomyia fasciata*, now *Aëdes aegypti*); to Walter Reed is due the credit of organizing the Board and directing the details of its work; to Surgeon General Sternberg for initiating the investigation; to Major General Leonard Wood who, as a medical man, appreciated the importance and vastness of the problem, as a friend of Reed did all in his power to assist, and as Governor-General gave official support to the measures proposed. Major Valery Havard, Chief Surgeon of the Cuba Division, directly instructed medical officers regarding mosquito control and other measures to prevent the spread of infection: Major W. C. Gorgas, Chief Sanitary Officer, by cleaning up Havana largely got rid of typhoid fever and "filth diseases," but was baffled by yellow fever till the mosquito theory of transmission was removed from theory to proven fact, when he again tackled the problem and learnt from it the lessons which he subsequently applied so successfully at Panama. And so on through the list to the more subsidiary but no less known actors in the drama; Major Kean who took over the duties of Colonel Havard when the latter went on leave; Captain Stark, Lazear, Pinto and others of the staff of the hospital at Colombia Barracks. All worked together in perfect harmony, discussing theories, devising and carrying out tests to prove or disprove them, assisting each and all in every conceivable way. Lastly, all honour is given to the volunteers who offered themselves for experiment, with exposure to fomites, and lent themselves to infection by blood or mosquito.

The book is well set up; the print good, illustrations excellent, and including the well-known picture by Dean Cornwell of "The Conquerors of Yellow Fever" as a frontispiece, with portraits of the chief actors, and later in the book a photograph of the Colombia Barracks Hospital Corps, including the volunteers, and, perhaps as historically instructive as any, a facsimile of Reed's letter to the author, dated Dec. 10th, 1900, probably the first account in writing of Reed's first experimental case of the disease. Misprints are very rare and there is only one peculiar *lapsus calami*, which a temporary *lapsus memoriae* on the part of the author calls a *lapsus pennae*—a pleasant touch of humour in a book the subject matter of which is necessarily sad, though the end was such a triumph—the story of him who "gave to man control over that dreadful scourge, Yellow Fever."

H. Harold Scott.

[Australian Military Forces.] **Friendly Fruits and Vegetables.** 71 pp., 37 figs. (11 coloured). Notified in G.O.'s dated 31st May 1943. Prepared by the General Staff, L.H.Q. Australia, and issued under the authority of the Commander Allied Land Forces, S.W.P.A. By Authority: Arbuckle Waddell Pty. Ltd., 20 McKillop St., Melbourne.

In this pamphlet descriptions are given, with plain and coloured photographs, of the many vegetables, fruits and nuts that are cultivated or grow wild in New Guinea and the adjacent islands. There are also notes on poisonous plants, hints on cooking, cleanliness and the care of cooking utensils, and on precautions in regard to water for drinking. The pamphlet is intended chiefly for the soldier who may become isolated in the jungle and have to depend on himself to keep alive. Most of the plants described are native to Australia so the soldier should receive practical instruction in their identification in the rear training areas. The list is a long one and includes such valuable and well known foods as cassava, maize, sweet potato, yam, banana, sugar cane, pawpaw, pineapple, mango, and coconut, as well as many with less familiar names. The "pidgin English" name and the local name are given in most cases. The book measures about 4 by 5 inches and weighs less than two ounces. *J. F. Corson.*

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Am.	signifies	Amoebiasis and Intestinal Protozoal Infections.
Bart.	,,	Bartonellosis.
Bl.	,,	Blackwater.
B.R.	,,	Book Review.
Chl.	,,	Cholera.
Der.	,,	Dermatology and Fungous Diseases.
Diet.	,,	Deficiency Diseases including Epidemic Dropsy.
Dys.	,,	Dysentery (Bacillary and Unclassed).
Fev.	,,	Fevers, including Dengue and Sandfly Fever.
Haem.	,,	Haematology.
Hel.	,,	Helminthiasis.
Leish.	signifies	Leishmaniasis.
Lep.	,,	Leprosy.
Mal.	,,	Malaria.
Misc.	,,	Miscellaneous.
Oph.	,,	Tropical Ophthalmology.
Pl.	,,	Plague.
Rab.	,,	Rabies.
R.F.	,,	Relapsing Fever and others Spirochaetoses.
Sp.	,,	Sprue.
Tryp.	,,	Trypanosomiasis.
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Y.F.	,,	Yellow Fever.
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Bregetova, N. G., with Monchadsky, Blagoveschensky & Ukhova, 1069 (Misc.)
Brent, R. H., with McCarthy, 214 (Fev.)
Brevdo, B. S., 660, 661 (Typh.)
Brewis, G. S., 7 (Mal.)
Briceño-Iragorry, L., 28, 391 (Typh.)
Briceño Rossi, A. L., 151 (Misc.)
—— & Iriarte, D. R., 866, 867 (Der.)
Bridges, R. F., 554 (Typh.)
Brink, V., 483 (Am.)
British Empire Leprosy Relief Association (Madras Provincial Council), 490 (Lep.)
British Guiana Med. Ann., 529 (Mal.)
British Medical Journal, 256, 357 (Mal.)
Brix, W., with Eyer, 23 (Typh.)
Broadbent, M. S. R., with Hussain, 359 (Mal.)
Brock, J. F., 142 (Diet.)
Brockbank, W. & Whittaker, S. R. F., 383 (Typh.)
Brocklebank, J. A., 861 (Hel.)
Brodie, B. B. & Udenfriend, S., 453 (Mal.)
Brodie, J., with Jamieson & Stiven, 754 (Dys.)
Broglie, with Schulten, 127 (Fev.)
Brookfield, R. W., with Findlay, 33 (Fev.)
Brown, H. R., Jr., Clark, W. F., Jones, N., Walther, J. & Warren, S. L., 241 (Misc.)
Brown, H. W., with Waletzky, 188 (Mal.)
Bruce, C. O., Knigin, T. D., Yolles, S. F. & Graham, A. E. Jr., 93 (Mal.)
Brug, S. L., with van der Meer, 152 (Misc.)
Brumpt, L. C., with Gallut, 848 (Chl.)
Brunner, M., Altman, I. & Bowman, K., 952 (Hel.)
Bryan, E. E., with Doull, 51 (Lep.)
Buchanan, J. A. C., 235 (Misc.)
Buchanan, R. N., with Turner, Snavely, Grossman & Foster, 750 (Y.F.)
Bugher, J. C., Boshell-Manrique, J., Roca-Garcia, M. & Osorno-Mesa, E., 475 (Y.F.)
—— & Gast-Galvis, A., 478 (Y.F.)
—— & Smith, H. H., 477 (Y.F.)
——, with —— & Bevier, 210 (Y.F.)
Bühler, F., 742 (Typh.)
Bull, G. M., with Berk, 145 (Haem.)
Bulletin de l'Institut d'Hygiène du Maroc, 233 (Misc.)
Bull. U.S. Army Med. Dept., 841 (Typh.), 860 (Hel.), 940 (Dys.)
Bulmer, E., 606 (Misc.)
de Burca, B., 640 (Mal.)

de Burca & Imdad Ali Shah, 640 (Mal.)
de Burgos Díaz-Varela, F., 547 (Tryp.)
Burhans, R. A., Camp, J. D., Butt, H. R. & Cragg, R. W., 599 (Hel.)
Burk, S. B. & Hodas, J. H., 220 (R.F.)
Burke, E., 469, 833 (Leish.)
Burnet, E., 1050 (Lep.)
—— & Cabasso, V., 1051 *bis* (Lep.)
——, ——, Cuénod, A. & Nataf, R., 1060 (Oph.)
Burroughs, A. L., 669 (Pl.)
——, with Meyer, Holdenried & Jawetz, 398 (Pl.)
Burrows, R., 408 (Hel.)
——, with Young, 219 (Am.)
Burton, A. F. & Kelsey, F. E., 260 (Mal.)
Buss, O., 154 (Misc.)
Bustamante, M. E. & Varela, G., 394 (Fev.)
Butler, F. A., 361 (Mal.)
Butt, H. R., with Burhans, Camp & Cragg, 599 (Hel.)
Buxton, P. A., 606, 782 (Misc.)

C.

Cabasso, V., with Burnet, 1051 *bis* (Lep.)
——, with ——, Cuénod & Nataf, 1060 (Oph.)
Caetano da Silva Júnior, J. A. & Flosi, A. Z., (501) (Hel.)
de Caires, P. F., 495 (Lep.)
Calcagno, O., with Fiol, 406 (Lep.)
Calderon, Barca, Rico-Avello & Rico, 200 (Fev.)
Camacho Téllez, J., Diaz Angulo, A., Romero Pérez, M. & Salazar, G., 203 (Typh.)
Cambournac, F. J. C., (976) (Misc.)
Cameron, E., 208 (Fev.)
Cameron, I. G., 260 (Mal.), 609 (Misc.)
Cameron, T. W. M., 761 (Hel.)
Camp, J. D., with Burhans, Butt & Cragg, 599 (Hel.)
Campos, J. A., 605 (Der.)
Campourcy, A., with Vaucel, 94 (Mal.)
Canby, C. B., Carpenter, G. & Ellmore, L. F., 961 (Haem.)
Cancela Freijo, J., with Alonso, (1059) (Der.)
Cançado, J. R., 223 (223) (Hel.)
Cannon, D. A., with Lesh, 134 (R.F.)
Canzani, R., with Claveaux, Salveraglio & Magnoni, 501 (Hel.)
Caplan, A., 239 (Misc.)
Cares, R., 363 (Mal.)
Carmichael, J. & Bell, F. R., 373 (Tryp.)
Carmona de la Fuente, A., 549 (Tryp.)
Carney, S. P. & Levin, N. B., 730 (Mal.)
Carpenter, C. M., Ackerman, H. & Ashenburg, N. J., 52 (Lep.)
Carpenter, G., with Canby & Ellmore, 961 (Haem.)
Carpenter, S. J., with Middlekauff, (781) (Misc.)
Carrick, L., 685 (Hel.)
Carson, D. A., 844 (Fev.)
Carter, C., with Heilman, Herrell & Greenburg, 1039 *bis* (R.F.)
Carter, C. L., 568 (Pl.)
Carter, R. H., Mann, H. D. & Smith, C. M., (617) (Misc.)
de Carvalho Pereira, M., with Rebêlo, (237) (Misc.)
Castañeda, R. M., 1018 (Typh.)
Castro Palomino, J., with Viamonte Cuervo, Alonso Suarez, Grau Triana & Romero Jordan, (1045) (Lep.)
Catanei, A., with Parrot, Collignon & Ambialet, 539 (Mal.)
Causey, O. R., Deane, L. M. & Deane, M. P., 644 (Mal.)
Cavanagh, J. R., 396 (Fev.)
Cawston, F. G., 947 (Hel.)
Ceccaldi, J., 178 (Rab.), 846 (Y.F.), 873 (Misc.)
Cerqueira, N. L., (781), (782) (Misc.)
——, with Lane, 155 (B.R.)
Chacon, R. V., with Mazza, 108 (Tryp.)
Chadwick, V., with Jones, Armstrong & Green, 773 (Diet.)
Chagas Filho C., 374, (929) (Tryp.)
Chakravarty, U. N. & Roy, S. C., (426) (Misc.)
Chalaya, L. E., 673 (Am.)
Chandler, A. C., with Bloxsom, 878 (Misc.)
Charters, A. D., 235 (Misc.)
Chatterjee, K. D., with Roy, (1006) (Mal.)
Chaudhuri, R. N., 45 (Am.), 517 (Misc.)
Chavira, C., with Mozzotti, 861 (Hel.)
Cheng, S. Y., with Hua, 419 (Diet.)
Chesterman, C. C., 606, 609 (Misc.), 789 (B.R.)
Chien, Y. C., with Li, 56 (Hel.)
Chifflet, A. & Peyrallo, R., (139) (Hel.)
Childress, M. E., with Kean, 50 (Lep.)
Chinen, S. S., with Hoagland & Harris, 577 (R.F.)
Chopra, B. L., 274 (Leish.)
Choqiette, L. P. E., with Bews, 573 (Am.)
Chorine, V., with Colas-Belcour, 758 (R.F.)
—— & Crougue, O., 574 (R.F.)
——, Grabar, P., Tixier, R. & Crougue, O., 291 (R.F.)
Chorley, J. K., 269 *bis*, 463 (Tryp.)
Choumara, R., 14 (Tryp.)
Chowdhury, N. K., with Prasad, 602 (Haem.)
Christie, J. R., *et al.*, 139 (Hel.)
Cimmino, V., 26 (Typh.)
Ciuca, M., Badenschi, G., Constantinescu, P. & Teriteanu, E., 645 (Mal.)
Clark, H. C., 9, (101), 924, (925), 1001 (Mal.)
Clark, W. F., with Brown, Jr., Jones, Walther & Warren, 241 (Misc.)
Clarke, A. G. & Prescott, 62 (Diet.)
Clastrier, J., with Parrot, (1012) (Leish.)
Claveaux, E., Salveraglio, F. J., Magnoni, P. & Canzani, R., 501 (Hel.)
Clavero, G. & Pérez Gallardo, F., 24 (Typh.)
Clearkin, P. A., 598 (Hel.)
Cleland, J. B., (236), 694 (Mal.)
Clemente, P., 177 (Rab.)
Coceani, A., 178 (Rab.)
Cochrane, E., 307 (Diet.)
—— & Newbold, C. E., 102 (Mal.)
Cochrane, R. G., 579 (Lep.)
——, with Fielding, 1048 (Lep.)
—— & Rajagopalan, G., 491 (Lep.)
Cockburn, T. A., with Findlay, 122 (Y.F.)
Côda, D., (915) (Mal.)
Coggeshall, L. T., 94 (Mal.)
——, with Maier, 826 (Mal.)

Cohen, H. G., with Witzberger, 942 (R.F.)
—— N. A., 279 (Fev.)
Colas-Belcour, J. & Chorine, V., 758 (R.F.)
Cole, A. C. E., 367 (Bl.), 831 (Leish.)
Collier, H. B. & Allen, D. E., 676 (Hel.)
Collier, H. O. J., with Lourie, 293 (R.F.), 370 (Tryp.)
Collignon, E., 101 (Mal.)
——, with Parrot, Catanei & Ambialet, 539 (Mal.)
Colonial Development and Welfare Act, 1940, (775) (Misc.)
Colonial Office, (233), (312) (Misc.)
Colonial Research Committee, (775) (Misc.)
Coluzzi, A., 353 *bis* (Mal.)
Colvin, S. H., Jr., Gore, I. & Peters, M., 774 (Der.)
Compton, A., 941 (Dys.)
Conant, N. F., & Howell, A., 67 (Der.)
Conge, M. & Boyer, F., 944 (R.F.)
Consolazio, F. C., with Johnson & Pitts, 974 (Misc.)
Consolazio, W. V., with Futcher & Page, 973 (Misc.)
Constantinescu, P., with Ciuca, Badenschi & Teriteanu, 645 (Mal.)
Cooley, R. A. & Kohls, G. M., (882) (Misc.)
Cooney, J. P. & Crosby, E. H., 584 (Lep.)
Cooper, H. K., with Randall, 1038 (R.F.)
Cooper, W. L., with Jackman, 283 (Am.)
Cooper, Z. K., with Moore & Weiss, 68 (Der.)
Cordero, E., with Peña Chavarria & Sáenz Herrera, 551 (Leish.)
Coria, J. B., with Gandolfo, Steinberg, Rugiero, Del Ponte & Crivellari, 311 (Der.)
Corrêa, R. R., 1006 (Mal.)
Costa, O. G., (232) (Der.), 737, 1011 (Leish.)
Cottrell, J. D., Stronach, R. D. & Peddie, J. J. G., 1033 (Fev.)
Covell, G., 646 (Mal.)
—— & Jaswant Singh, 455 (Mal.)
Cox, W. W., with Eyles, 642 (Mal.)
Cragg, R. W., with Burhans, Camp & Butt, 599 (Hel.)
Craig, C. F. & Faust, E. C., 521 (B.R.)
Crawford, P. J., with Kumm, 123 (Y.F.)
Crawford, T., 1002 (Mal.)
Crawford, W., 1041 (Lep.)
Crilly, J. A., with Krause, 139 (Hel.)
Critchley, M., with Allison, 519 (Misc.)
Crivellari, C. A., with Steinberg, 912 (Rab.)
Crivellari, E., with Gandolfo, Steinberg, Rugiero, Del Ponte & Coria, 311 (Der.)
Crofton, J. & Dick, J. C., 1012 (Typh.)
Crosby, E. H., with Cooney, 584 (Lep.)
Crougue, O., with Chorine, 574 (R.F.)
——, with ——, Grabar & Tixier, 291 (R.F.)
Cruveilhier, L. & Viala, C., 178 (Rab.)
Cuénod, A., with Burnet, Cabasso & Nataf, 1060 (Oph.)
Cuff, J. R., with Turner, Bent, Holloway & Quinland, 148 (Misc.)
Culbertson, J. T. & Rose, H. M., 772 (Hel.)
——, —— & Demarest, C. R., 770, 771 (Hel.)
Cullinan, E. R. & Whittaker, S. R. F., 126 (Fev.)
da Cunha, J. F., with Fox & Kossobudski, 123 (Y.F.)
Cunningham-van Someren, G. R., 1006 (Mal.)
Curd, F. H. S., 101 (Mal.)
Cutkomp, L. K., 320 (Misc.)
Czertok, J., with Braun & Kornblueth, 186 (Mal.)

D.

Daggett, W. I., 318 (Misc.)
Daniels, W. B. & Grennan, H. A., 278 (Fev.)
——, with Roberts, 41 (Dys.)
Dao, L., 96 (Mal.)
Das, B. K., 89 (Mal.)
Das Gupta, B. M. & Siddons, L. B., 11, 99, 545, 826 (Mal.)
Das Gupta, C. R., 92 (Mal.)
—— & Sen Gupta, P. C., 468 (Leish.)
Daver, M. B. & Ahmed, S. S., 21 (Leish.)
Davey, D. G., 365 (Mal.)
Davey, T. F., 53 (Lep.)
—— & Ross, C., 587 (Lep.)
David, N. A., Phatak, N. M. & Zener, F. B., 672 (Am.)
David, W. A. L., 664 (Typh.)
—— & Bracey, P., 880 (Misc.)
Davies, R. A., 448 (Mal.)
Davis, D. H. S., with Gear & de Meillon, 739 (Typh.)
Davis, D. J., 271 (Tryp.)
Davis, E. L., 508 (Der.)
Davis, G. E., 32 *bis*, 393 *bis* (Fev.), 46 (R.F.)
Davis, H., 1052 (Hel.)
Davis, W. A., Juvera, F. M. & Lira, P. H., 744 (Typh.)
—— & Wheeler, C. M., 743 (Typh.)
Dawber, T. R., 364 (Mal.)
Deane, L. M., with Causey & Deane, M. P., 644 (Mal.)
Deane, M. P., with Causey & Deane, O. R., 644 (Mal.)
Dearborn, E. H., with Kelsey, Geiling & Oldham, 923 (Mal.)
De La Lastra Soubrier, J. M., 470 (Typh.)
DeLanney, L. A. & Beahm, E. H., 486 (Am.)
Dellaert, R., with Rodhain, 544 (Mal.)
De Los Angeles Sandoval, M., with González Ochoa, 70 (Der.)
Del Ponte, E., with Gandolfo, Steinberg, Rugiero, Crivellari & Coria, 311 (Der.)
Del Sel, M., 851 (Am.)
Demarest, C. R., with Culbertson & Rose, 770, 771 (Hel.)
Demianov, E. A., 486 (Am.)
Denhoff, E. & Piper, B. C., 918 (Mal.)
Denison, N., 467 (Tryp.)
Dennison, W. M. & Dick, A. L., 776 (Misc.)
Derrick, E. H., 558 (Fev.)
Deschiens, R. & Lamy, L., (1056) (Hel.)
Devignat, R., 668 (Pl.)
Devine, J., 613 (Misc.), 930 (Leish.)
Dharmendra, (53), 584, 585, 1050 (Lep.)
—— & Bose, R., 588 (Lep.)
—— & Jaikaria, S. S., 50 (Lep.)
—— & Santra, I., 1045 (Lep.)
Dhayagude, R. G., (865) (Haem.)
—— & Purandare, N. M., 533 (Mal.)
Diaz Angulo, A., with Camacho Téllez, Romero Pérez & Salazar, 203 (Typh.)
Diaz-Rivera, R. S., with Kirsh, 218 (Am.)
—— & Rasberry, E. A., 1036 (Am.)

Dick, A. L., with Dennison, 776 (Misc.)
Dick, J. C., with Crofton, 1012 (Typh.)
Dickson, J. G., Huntington, R. W., Jr. & Eichhold, S., 303 (Hel.)
Dillenberg, H., with Eyer, 657 (Typh.)
Dinan, J. F., with Faget, Pogge, Johansen, Prejean & Eccles, 494 (Lep.)
Ding, E., 116 (Typh.)
Diniz, O., 1057 (Diet.)
Diomina, N. A., with Mochkovski, Nossina, Pavlova, Livchitz, Pels & Roubtzova, 565 (Fev.)
Dios, R. L. & Bonacci, H., 468 (Tryp.)
—— & de Sommerville, E. T. W., 829 (Tryp.)
Dixon, K. C., 46, 219 (R.F.), 723 (Mal.)
Dobell, C., 45 (Am.)
Dobes, W. L., 69 (Der.)
Dockerty, M. B., with Jacobson, Jr., 231 (Der.)
Dominguez Sisco, R., 855 (Y. & S.)
Don, C. S. D. & Meyer, P. F., 358 (Mal.)
Donaldson, A. W., 141 (Hel.)
Dormer, B. A., Friedlander, J. & Wiles, F. J., 519 (Misc.)
Douglass, E. D., with Wikler, Williams & Emmons, (70) (Der.)
Doull, J. A. & Bryan, E. E., 51 (Lep.)
——, Guinto, R. S., Rodriguez, J. N. & Bancroft, H., 48 (Lep.)
Downs, W. G., Gillette, H. P. S. & Shannon, R. C., 445 (Mal.)
Doyle, W. L., 151 (Misc.)
Drew, R., 782 (Misc.)
Dreyfuss, F., 573 (Am.)
—— & Gurevitch, J., 568 (Dys.)
Druckmann, A. & Schorr, S., 850 (Am.)
Dubois, A., 494 (Lep.)
Duguid, J. B. & Sheppard, E. M., 857 (Hel.)
Dukhanina, N. N., with Levenson, Fastorskaya & Khovanskaya, 10 (Mal.)
Dunlap, G. L., 176 (Rab.)
Duren, A., 56 (Hel.)
Dyer, R. E., (739) (Fev.)

E.

Eads, J. T. & Kash, R. M., 236 (Misc.)
Eads, R. B., with McGregor, (617) (Misc.)
Eagle, H. & Magnuson, H. J., with Musselman, A. D., 942 (R.F.)
Earle, K. V., 768 (Hel.)
Earnshaw, H., 509 (Der.)
East African Med. J., 229 (Vms.), 299 (Hel.)
Eaton, M. D., with Sawyer, Meyer, Bauer, Putnam & Schwentker, 1026 (Y.F.)
Eddy, G. W., with Joyce, 618 (Misc.)
Eccles, C. G., with Faget, Pogge, Johansen, Dinan & Prejean, 494 (Lep.)
Eckardt, P., 277 (Fev.)
Eckstein, A., 361 (Mal.)
Edge, P. G., 727 (Mal.), 977 (B.R.)
Edgett, R., with Manwell, 189 (Mal.)
Egnarian, V. I., with Oganesov, Sadoyan, Istamanian & Karapetian, 535 (Mal.)
Eichhold, S., with Dickson & Huntington, 303 (Hel.)
Eichna, L. W., with Bean, 1071 (Misc.)
Eigenfeld, D. D. & Schlesinger, C. J., 496 (Hel.)
Ellis, F. A., 882 (Misc.)
Ellmore, L. F., with Canby & Carpenter, 961 (Haem.)
Elmes, B. G. T. & Hall, R. N., 833 (Leish.)
Elsdon-Dew, R., 486 (R.F.)
van Emden, F. I., 829 (Tryp.)
Emerson, K., Jr., 425 (Misc.)
Emmons, C. W., with Wikler, Williams & Emmons, (70) (Der.)
Encinas, E., with Bernales, 58 (Hel.)
English, R. B., 507 (Haem.)
Enright, J. R., 563 (Fev.)
Epstein, E. F., with Tokarevich, 1017 (Typh.)
Epstein, E. G., 451 (Mal.)
Ernst, K. & Portius, W., 559 (Fev.)
Espin, J., with Vegas, 581 (Lep.) *bis*
Evans, H. M., 884 (B.R.)
Evans, K. L., with Plotz, Woodward & Philip, 28 (Typh.)
Evans, R. W., 505 (Haem.)
Evans, W., 146 (Haem.)
Eyer, H. & Brix, W., 23 (Typh.)
—— & Dillenberg, H., 657 (Typh.)
Eyles, D. E., 642 (Mal.)
—— & Cox, W. W., 642 (Mal.)

F.

Faget, G. H., 296 (Lep.)
—— & Johansen, F. A., 51 (Lep.)
—— & Mayoral, A., 583 (Lep.)
——, Pogge, R. C., Johansen, F. A., Dinan, J. F., Prejean, B. M. & Eccles, C. G., 494 (Lep.)
—— & Ross, H., 1046 (Lep.)
Fairbairn, H., 12 (Tryp.)
Fairchild, G. B., 429 (Misc.)
Fales, J. H., with McGovran, 617 (Misc.)
Fastorskaya, E. I., with Levenson, Khovanskaya & Dukhanina, 10 (Mal.)
Fauconnier, J., with Berge & Audoye, 664 (Typh.)
——, with Laurent, Barge, Berge & Audoye, 760 (Hel.)
Faust, E. C., 91, 998 (Mal.), 670, (758) (Am.), 966, 967 (Misc.)
——, with Craig, 521 (B.R.)
Fawcett, D. W. & Gens, J. P., 518 (Misc.)
Fawdry, A. L., 504 (Haem.)
Fay, H. B., & Johnston, C. A. W., 216 (Am.)
Fehily, L., 142 (Diet.)
Feldman-Muhsam, B., (696) (Misc.)
Felix, A., 552 (Typh.), 606, 609 (Misc.)
Fenton-Russell, D., 921 (Mal.)
Ferguson, J. W., (143) (Diet.)
Ferguson, R. L., 127 (Fev.)
Ferguson, W. J. W., 687 (Diet.)
Fernandez Marina, R., 1004 (Mal.)
Fernández Vautrei, R., 579 (Lep.)
Fernando, P. B. & Balasingham, S., 413 (Hel.)
Ferrari, J., with Sordelli, Manzullo & Riessel, 388, 390 *ter*, 391 (Typh.)
Ferro-Luzzi, G., 194 (Leish.)
Ferry, W., with Gaugier, 62 (Diet.)
Fiasson, R., 695 (Misc.)

Fielding, J. W., 24 (Typh.), 1049 (Lep.)
—— & Cochrane, R. G., 1048 (Lep.)
Findlay, G. M., 105 (Bl.)
—— & Brookfield, R. W., 33 (Fev.)
—— & Cockburn, T. A., 122 (Y.F.)
——, Martin, N. H. & Mitchell, J. B., 1028 (Y.F.)
Finlayson, M. H., 776 (Misc.)
——, with Mayer, 694 (Misc.)
Fiol, H., 407 (Lep.)
—— & Blanco, J. F., 494 (Lep.)
—— & Calcagno, O., 406 (Lep.)
—— & Zambrano, J., 406 (Lep.)
Firmino Sant'anna, J., 515 (Misc.)
Fischer, L., 313 (Misc.)
Fitz-Hugh, T., Jr., with Livingood & Rogers, 230 (Der.)
Flosi, A. Z., with Caetano da Silva Júnior, (501) (Hel.)
Flynn, P. D., 956 (Hel.)
Fogel, R. H., with Rome, 304 (Hel.)
Foley, E. J., 859 (Hel.)
da Fonseca, F., with da Fonseca, J. A. B., 93 (Mal.)
da Fonseca, J. A. B. & da Fonseca, F., 93 (Mal.)
Font, J. H., 302 (Hel.)
Fornieles Ulibarri, F., (1069) (Misc.)
Forrest, G. H., 1067 (Misc.)
Foster, S. O., with Turner, Snavely, Grossman & Buchanan, 750 (Y.F.)
Fox, F. W., 226 (Diet.)
Fox, H., 518 (Misc.)
Fox, J. P., Kossobudzki, S. L. & da Cunha, J. F., 123 (Y.F.)
—— & Penna, H. A., 125 (Y.F.)
Foy, H., Gluckman, J. & Kondi, A., 651 (Bl.)
—— & Kondi, A., 603 (Haem.)
Fradkina, R. V., with Smorodintzeff, 1021 (Typh.)
Fraga, J., 447 (Mal.)
Francis, E., 214 (Pl.)
Francis, J., 136 (Lep.)
Frank, H. R., with Alwens, 274 (Typh.)
Frank, J. R., 572 (Am.)
Frazier, C. N., 579 (Lep.)
Freeborn, S. B., 729 (Mal.)
French, M. H., with Hornby, 107 (Mal.)
Friedlander, J., with Dormer & Wiles, 519 (Misc.)
Friedlander, L., with Gillman, T., Gillman, J., Inglis & Hammar, 1057 (Diet.)
Fróes, H. P., 125, (213) (Y.F.)
Fulton, F., with Begg & van den Ende, 838 (Typh.)
Fulton, J. D., 377, 929 (Tryp.)
—— & Yorke, W., 15, 373 (Tryp.)
Funke, A., Bovet, D. & Montezin, G., 734 (Tryp.)
Furnell, M. J. G., 865 (Der.)
Futcher, P. H., Consolazio, W. V. & Page, N., 973 (Misc.)

G.

Gaase, 114 (Fev.), 378, 472 (Typh.)
Gaehtgens, W., 138 (Hel.)
Gallia, F., with Kubes, 905 (Rab.)
Gallut, J. & Brumpt, L. C., 848 (Chl.)
—— & Grabar, P., 401, 402 (Chl.)
Galperin, E. P., 825 (Mal.)
Galton, E. M. G., 1061 (Oph.)
Galvão, A. L. A., (917) (Mal.)
——, Lane, J. & Unti, O., (917) (Mal.)
Gamal El Din El Hefny, A., with Kamal, 566 (Pl.)
Gambia, 106 (Tryp.), 315, 874 (Misc.)
Gammie, R. P., 1002 (Mal.)
Gandolfo, C. F., Steinberg, I. R., Rugiero, H. R., Del Ponte, E., Crivellari, C. & Coria, J. B., 311 (Der.)
Ganguli, H. D., with Bagchi, 428 (Misc.)
Garcia Barriga, H., (587) (Lep.)
Gard, J. J., 282 (Dys.)
Gardiner, P. A., with Lyle & Macrae, 688 (Diet.)
Garland, L. H., 147 (Misc.)
Garry, T. G., 702 (B.R.)
Garzon, R. & Pitt, L. A., 135 (Lep.)
Gast Galvis, A., (449) (Mal.)
——, with Bugher, 478 (Y.F.)
Gates, J. G., (1036) (Pl.)
Gathy, J., with Appelmans, 519 (Misc.)
Gaugier, P. & Ferry, W., 62 (Diet.)
Gauwerky, F., with Pfeffer, 274 (Typh.)
Gavan Duffy, C. A., (921) (Mal.)
Gayed, I., with Kamal & Anwar, 566 (Pl.)
Gay Prieto, J., 586 (Lep.)
Gear, J., de Meillon, B. & Davis, D. H. S., 739 (Typh.)
Gear, J. H. S., Yeo, R. M. & Bodenstein, J. C., 1067 (Misc.)
Geiger, J. C., with Reed, 886 (B.R.)
Geiling, E. M. K., with Kelsey & Oldham, 11 (Mal.)
——, with ——, —— & Dearborn, 923 (Mal.)
Geiman, Q. M., (12) (Mal.)
Gelfand, M., 318 (Misc.)
—— & Osburn, H. S., 137 (Hel.)
Genatios, T., with Alarcón, 614 (Misc.)
Gens, J. P., with Fawcett, 518 (Misc.)
Gent, J. C., 6 (Mal.)
Germer, W. D. & Behrens, H., 429 (Misc.)
Ghosh, B. K., with Mukerji & Siddons, 9 (Mal.)
Ghosh, B. N. & Adhya, P. C., 289 (Am.)
Ghosh, L. M., with Panja, 614 (Misc.)
Ghosh, S. K., with Panja, 40 (Chl.)
Ghosh, S. M., with Roy, 972 (Misc.)
Gibbons, R. J., 392 (Fev.)
Giblin, W. E., 689 (Haem.)
Gibson, M. O. J., with Lipscomb, 550 (Leish.)
Giffen, H. K., with Saunders, 50 (Lep.)
Gil Bermúdez, A., 655 (Leish.)
Gilbert, B., 296 (Hel.), 427 (Misc.)
Gilchrist, B. M., with Bishop, 731 (Mal.)
Gillett, J. A., 538 (Mal.)
Gillette, H. P. S., with Downs & Shannon, 445 (Mal.)
Gillman, J., with Gillman, T., Inglis, Friedlander & Hammar, 1057 (Diet.)
Gillman, T. & Gillman, J., 1058 (Diet.)
——, ——, Inglis, J., Friedlander, L. & Hammar, E., 1057 (Diet.)
Gilmore, R. M., 211 (Y.F.)
Giorgi, D., with Mendes de Castro & Kieffer, 522 (B.R.)

Girard, G., 36 (Pl.)
——, with Roubaud, 567 (Pl.)
Giraud, P. & Revol, P., 109 (Leish.)
Giroud, M. L., with Giroud, P. & Meunier, 738 (Typh.)
Giroud, P., Giroud, M. L. & Meunier, M., 738 (Typh.)
Giuntini, J., with Lépine, Levaditi & Grabar, 913 (Rab.)
Glasgow, J. P. & MacInnes, D. G., 6 (Mal.)
Glazier, M. & Olson, C., 1070 (Misc.)
Glück, G., 576 (R.F.)
Gluckman, J., with Foy & Kondi, 651 (Bl.)
Goder, G. A., with Heldt, 258 (Mal.)
Goldsmith, N. R., 231 (Der.)
Gontaeva, A. A., 280 (Fev.)
Gontayeva, A. A., with Tarejev & Rotenburg, 257 (Mal.)
González, E., 931 (Typh.)
Gonzalez Guzman, I., 404 (Lep.)
González Ochoa, A. & De Los Angeles Sandoval, M., 70 (Der.)
Gonzalez Prendes, M. A., with Ibarra Perez, 760, (1045) (Lep.)
Goobar, J. K., with de Villafañe Lastra, Rodeiro & Videla, 400 (Pl.)
——, with —— & ——, 398, 400 (Pl.)
Goodenough, W., (884) (Misc.)
Goodhue, L. D., (972) (Misc.)
Goodman, J. I., with Wolkin & Kelley, 785 (Misc.)
Goodwin, L. G. & Page, J. E., 20 (Leish.), 222 (Hel.)
Goodwin, M. H., Jr. & Lenert, L. G., 183 (Mal.)
Gore, I., with Colvin & Peters, 774 (Der.)
Gorgas Memorial Laboratory Ann. Rep., 9, 924 (Mal.)
Gottlieb, A., (965) (Der.)
Gougis, R., with Parrot, 736, (1012) (Leish.)
Grabar, P., with Chorine, Tixier & Crougue, 291 (R.F.)
——, with Gallut, 401, 402 (Chl.)
——, with Lépine, Levaditi & Giuntini, 913 (Rab.)
Grace, A. W., 427 (Misc.)
Graham, A. E., Jr., with Bruce, Knigin & Yolles, 93 (Mal.)
Graña, A., 764 (Hel.)
Grant, G. H., with Apley, 1065 (Misc.)
Gratch, I., 197 (Fev.)
Grau Triana, J., with Viamonte Cuervo, Alonso Suarez, Castro Palomino & Romero Jordan, (1045) (Lep.)
Green, A. A., with Parkin, (187) (Mal.), 880 (Misc.)
Green, H. F., with Jones, Armstrong & Chadwick, 773 (Diet.)
Greenburg, N., with Heilman, Herrel & Carter, 1039 *bis* (R.F.)
Greenwood, M., 977 (B.R.)
Gregory, J. R., 733 (Bl.)
Greiff, D., with Moragues & Pinkerton, 839 (Typh.)
—— & Pinkerton, H., 840 (Typh.)
Greig, E. D. W., 291 (R.F.)
Grenier, P., with Roubaud, 881 (Misc.)
Grennan, H. A., with Daniels, 278 (Fev.)
Grindlay, J. H., 777 (Misc.)
Grosperrin & Mauric, 78 (Misc.)
Grossman, E. B., with Turner, Snavely, Buchanan & Foster, 750 (Y.F.)
Gudex, T. V., with Simpson, Leake, McMahon & Rueckert, 359 (Mal.)
Guimarães, F. N., 321 (Misc.)
Guimarães, R., with de Magalhães, 228 (Vms.)
Guinto, R. S., with Doull, Rodriguez & Bancroft, 48 (Lep.)
——, with Saunders, 49 (Lep.)
Gundel, M. E., with Seeler & Ott, 651 (Mal.)
Gurd, D. P., 509 (Oph.)
Gurevitch, J., 46 (Am.)
——, with Dreyfuss, 568 (Dys.)
Gurney, A. B., (617) (Misc.)

H.

Habbu, M. K., with Sokhey, 129 *bis* (Pl.)
Haber, H., 1041 (Lep.)
Hackett, L. W., 998 (Mal.)
Hailey, (233) (Misc.)
Hall, R. N., with Elmes, 833 (Leish.)
Hall, T. F., with Hess, 695 (Misc.)
Halliday, J. H. & Horan, J. P., 279 (Fev.)
Hamann, C. B., 415 (Hel.)
Hamburger, H. J., 723 (Mal.)
Hamilton, C. R., with Sandground, 372 (Tryp.)
Hamlin, M. T., with Scudi, 262, 263, 728 (Mal.)
Hammar, E., with Gillman, T., Gillman, J., Inglis & Friedlander, 1057 (Diet.)
Hammerstrom, R. J., with Sawitz, 43 (Am.)
Hanschell, H. M., 71 (Misc.)
Hardikar, S. W. & Rao, V. G., 319 (Misc.)
Harding, R. D., 1009 (Tryp.)
Harford, C. G., with Sulkin, 171 (Rab.)
Harley, R. D., with Snow, (774) (Der.)
Harper, J. O., 821 (Mal.)
Harris, F. H., with Hoagland & Chinen, 577 (R.F.)
Harris, J. R., with Hickey, 858 (Hel.)
Harris, S. & Harris, S., Jr., 307 (Diet.)
Hartz, P. H., 955 (Hel.)
Hase, A., 386 (Typh.)
Hauer, A., 8 (Mal.)
Hawes, R. B., 609 (Misc.)
Hawking, F., 71 (Misc.), 732, 1005 (Mal.), 854 (R.F.)
Hazen, E. L., with Maillard, 474 (Fev.)
Hecht, O., 1034 (Pl.)
Hegsted, D. M., McKibbin, J. M. & Stare, F. J., 649 *bis* (Mal.)
Heilig, R. & Puttaya, M., 428 (Misc.)
——, with Topping & Naidu, 29 (Typh.)
—— & Visveswar, S. K., 285 (Am.), 499 (Hel.), 615 (Misc.)
Heilman, F. R. & Herrell, W. E., 758 (R.F.)
—— & ——, with Carter, C. & Greenburg, N 1039 *bis* (R.F.)
Heimberger, 384, 556 (Typh.), 823 (Mal.)
Heinrich, K., with Kuhlmann, 932 (Typh.)
Heldt, T. J. & Goder, G. A., 258 (Mal.)
Hemming, J. R., with Seelig, 724 (Mal.)
Henderson, T. L. C., 307 (Diet.)
Hennessey, R. S. F., 320 (Misc.)

Henrard, C., with van Hoof & Peel, 928 (Tryp.)
——, with Wanson, (617) (Misc.)
Henry, A. J., 196 (Leish.)
Henschel, A. F., with Taylor & Keys, 322, 882 (Misc.)
——, with ——, Mickelsen & Keys, 883 (Misc.)
Herbert, P. H., with Beattie, Wechtel & Steele, 409 (Hel.)
Hermitte, L. C. D., 183 (Mal.)
Hermosilla Diaz, F., Awad, S. & Rojas, M., (735) (Tryp.)
Herms, W. B., (972) (Misc.)
Herrell, W. E., with Heilman, 758 (R.F.)
——, with ——, Carter & Greenburg, 1039 *bis* (R.F.)
Herrera, J. A., with Baliña, Bosq & Barnatán, 421 (Der.)
——, with ——, —— & Negroni, 421 (Der.)
Herrlich, A., (218) (Am.)
—— & Liebmann, H., 782 (Misc.)
Hertig, M., (110) (Leish.)
——, with Howe, 563 (Bart.)
Hess, A. D. & Hall, T. F., 695 (Misc.)
Hesse, E. & Kremser, H., 1024 (Fev.)
Hewitt, R. I., (926) (Mal.)
—— & Richardson, A. P., 188 (Mal.)
Hickey, M. D. & Harris, J. R., 858 (Hel.)
Hill, R. B., with Pitta Simões, 496 (Hel.)
Hillman, C. C., 871 (Misc.)
Hirshfeld, J. W., with Noth, 757 (Am.)
Hirvonen, M., 594 (Hel.)
Hitch, J. M., 867 (Der.)
Hitzig, W. M. & Liebesman, A., 943 (R.F.)
Hixson, H., 532 (Mal.)
Hoagland, R. J., Harris, F. H. & Chinen, S. S. 577 (R.F.)
Hoare, C. A., 150 (Misc.)
Hodas, J. H., with Burk, 220 (R.F.)
von Hofe, F. H., 958 (Hel.)
Hoff, F., 661 (Typh.)
Holden, J. R., 548 (Tryp.)
Holdenried, R., with Meyer, Burroughs & Jawetz, 398 (Pl.)
Holinger, P. H., with van Pernis & Benson, 70 (Der.)
Hollaender, A., with Jones, 596 (Hel.)
Hollis, M. D., (1006) (Mal.)
Holloway, G. D., with Turner, Bent, Cuff & Quinland, 148 (Misc.)
Holman, E. & Pierson, P., 762 (Hel.)
Hompesch, H., 137 (Hel.)
Hood, M., 132 (Am.)
Hopp, W. B., 879 (Misc.)
Horan, J. P., with Halliday, 279 (Fev.)
Horn, D. W., 315 (Misc.)
Hornby, H. E. & French, M. H., 107 (Tryp.)
Horrenberger, R., with Benhamou & Renoux, 25 (Typh.)
Horst, 215 (Am.)
Horvath, S. M., with Robinson, Turrell & Belding, 520 (Misc.)
we, C., 209 (Bart.)
—— & Hertig, M., 563 (Bart.)
Howell, A., with Conant, 67 (Der.)
Howie, J. W., 71 (Misc.)
—— & Murray-Lyon, R. M., 185 (Mal.)
Hua, T. J. & Cheng, S. Y., 419 (Diet.)
Huang, J., 849 (Chl.)
Hudson, E. H., (613), 875 (Misc.)
Huff, C. G., 619 (B.R.)
——, with Thompson, 827 *bis* (Mal.)
Huffaker, C. B. & Back, R. C., 237 (Misc.)
Hughes, J. P. W., (616) (Misc.)
Hughes, S. B. & Bomford, R. R., 357 (Mal.)
Hughes, W., 670 (Dys.)
Hunter, J., with Pick, 924 (Mal.)
Huntington, R. W., Jr., with Dickson & Eichhold, 303 (Hel.)
Hurlbut, H. S., 266 (Mal.)
Hussain, K. K. & Broadbent, M. S. R., 359 (Mal.)
Hutter, A. M., with Bozicevich, 1055 (Hel.)
Hyman, A. S., 397 (Fev.)

I.

Ibarra Perez, R. & Gonzalez Prendes, M. A., 760, (1045) (Lep.)
Ibrazimova, N. V., with Shukov-Vereshnikov, Kolodeznaja & Zaletaev, 741 (Typh.)
Ignacio Chala, J. & Lleras Restrepo, F., 587 (Lep.)
Imdad Ali Shah, with de Burca, 640 (Mal.)
Indian Medical Gazette, 18 (Leish.), 287 (Am.), 517 (Misc.)
Indian Research Fund Association, 38 (Pl.), 39 *bis* (Chl.), 47, 51 (Lep.)
Inglis, J., with Gillman, T., Gillman, J., Friedlander & Hammar, 1057 (Diet.)
Inter-American Co-operative Health Services, 722 (Mal.)
Ionina, N. C., 499 (Hel.)
Iriarte, D. R., 66 (Der.)
——, with Briceño Rossi, 866, 867 (Der.)
Irvine, C., 878 (Misc.)
Istamanian, L. S., with Oganesov, Sadoyan, Egnarian & Karapetian, 535 (Mal.)
Iturbe, J., 854 (Y. & S.)
Ivanhoe, G. L., 132 (Am.)
Ivanissevich, O., Taiana, J. A., Niño, F. & Rosito, E., 284 (Am.)
Ivy, A. C., with Annegers, Snapp, Paskind & Atkinson, 98 (Mal.)

J.

Jachowski, L., Jr., 37 (Pl.)
Jackman, R. J. & Cooper, W. L., 283 (Am.)
Jacobs, H. R., 458 (Mal.)
Jacobs, L., with von Brand, Rees & Reardon, 133 (Am.)
——, with Wright & Kerr, 502 (Hel.)
Jacobson, C. F., Jr. & Dockerty, M. B., 231 (Der.)
Jadin, J., 1015 *bis* (Typh.)
Jaffé, R., with K. Neubürger, 1057 (Diet.)
Jaikaria, S. S., with Dharmendra, 50 (Lep.)
James, S. P., 71, 782 (Misc.)
Jamieson, W. M., Brodie, J. & Stiven, D., 754 (Dys.)
Jana, A. P., 568 (Dys.)
Jaramillo, J. R., 746 (Bart.)
Jaswant Singh, with Covell, 455 (Mal.)
Jauregui, R. B., with Mazza, 375 (Tryp.)

Jawetz, E. & Meyer, K. F., 400, 753, 847, 1034, 1035 (Pl.)
——, with ——, Holdenried & Burroughs, 398 (Pl.)
Jelinek, V. C., with Scudi, 452 (Mal.)
——, with —— & Kuna, 728 (Mal.)
Johannessohn, F., 727 (Mal.)
Johansen, F. A., with Faget, 51 (Lep.)
——, with ——, Pogge, Dinan, Prejean & Eccles, 494 (Lep.)
Johnson, C. E., Jr., 100 (Mal.)
Johnson, F. H. & Schneyer, L., 923 (Mal.)
Johnson, H. N., 179 (Rab.)
Johnson, R. E., 238 (Misc.)
——, Pitts, G. C. & Consolazio, F. C., 974 (Misc.)
Johnston, C. A. W., with Fay, 216 (Am.)
Jones, H. E., Armstrong, T. G., Green, H. F. & Chadwick, V., 773 (Diet.)
Jones, J. W., Jr., 532 (Mal.)
Jones, M. F. & Hollaender, A., 596 (Hel.)
Jones, N., with Brown, Jr., Clark, Walther & Warren, 241 (Misc.)
Jones, T. C. & Maurer, F. D., 937 (Y.F.)
Jordan, K., 328 (B.R.)
Jörg, M. E., with Mazza, (830) (Tryp.)
——, with ——, Basso, G., Basso, R. & Miyara, 16 (Tryp.)
Jorstad, L. H., with Moore, 691 (Der.)
Joubert, J. D., 693 (Misc.)
Journal of the American Medical Association, 96 (Mal.), 692 (Misc.)
Journal of the National Malaria Society, 996 (Mal.)
Journal of Parasitology, (997 (Mal.)
Joyce, C. R. & Eddy, G. W., 618 (Misc.)
Jukov, N. M. & Krasikova, V. I., 917 (Mal.)
Junior, M. da S., (39) (Pl.)
Jurány, E., with Lörincz, 219 (Am.)
Juvera, F. M., with Davis & Lira, 744 (Typh.)

K.

Kahler, O. H., 451 (Mal.)
Kaiser, M., 911 (Rab.)
Kalandadze, L. P. & Sagatelova, I. S., 916 (Mal.)
Kallat, S., 940 (Pl.)
Kalz, F. & Newton, B. L., 488 (Y. & S.)
Kamal, A. M., 566 (Pl.)
—— & Gamal El Din El Hefny, A., 566 (Pl.)
——, Gayed, I. & Anwar, M., 566 (Pl.)
Kamal, A. M. & Messih, G. A., 380, 382 (Typh.)
Karapetian, R. N., with Oganesov, Sadoyan, Istamanian & Egnarian, 535 (Mal.)
Kark, E., 1066 (Misc.)
Kark, S. L., 418 (Diet.), 611 (Misc.)
—— & Le Riche, H., 775 (Misc.)
Karlen, M. A., 285 (Am.)
Kash, R. M., with Eads, 236 (Misc.)
Kathe, J. & Peters, F., (142) (Hel.)
Katsampes, C. P., McCoord, A. B. & Phillips, W. A., 852 (Am.)
Kay, H. B., with Bonnin, 282 (Dys.)
Kean, B. H., 149 (Misc.)
—— & Childress, M. E., 50 (Lep.)
Keilin, J., 1008 (Bl.)
Kekhcher, O. M., 258, 449 (Mal.)
Kellert, E., 236 (Misc.)
Kelley, W. E., with Wolkin & Goodman, 785 (Misc.)
Kelsall, A. R., with Leishman, 1062 (Misc.)
Kelsey, F. E., with Burton, 260 (Mal.)
——, Geiling, E. M. K., Oldham, F. K. & Dearborn, E. H., 923 (Mal.)
—— & Oldham, F. K., 260 (Mal.)
——, with ——, 260 (Mal.)
——, —— & Geiling, E. M. K., 11 (Mal.)
Kemper, J. W. & Bloom, H. J., 964 (Der.)
Keogh, P. & Shaw, F. H., 365, 1003 (Mal.)
Kern, B. & Kustner, H., 644 (Mal.)
Kerr, K. B., with Wright & Jacobs, 502 (Hel.)
Kevorkov, N. P., 498 *bis* (Hel.)
Kevorkova, V. I., with Khodukin & Soshnikova, 938 (Fev.)
Keys, A., with Henschel & Taylor, 882 (Misc.)
——, with Taylor & Henschel, 322 (Misc.)
——, with ——, —— & Mickelsen, 883 (Misc.)
Keys, T. E., (568) (Pl.)
Khan, F. K., 228 (Vms.)
Khartoum, 422 (Misc.)
Khodukin, N. I. & Shterngold, E. J., 939 (Fev.)
——, Soshnikova, M. N. & Kevorkova, V. I., 938 (Fev.)
Khovanskaya, A. I., with Levenson, Fastorskaya & Dukhanina, 10 (Mal.)
Kieffer, J. C., with Mendes de Castro & Giorgi, 522 (B.R.)
Kikuth, W., (470) (Leish.), 724 (Mal.)
—— & Schmidt, H., 194 (Leish.)
Killian, H. & Obertreis, E., 662 (Typh.)
Kinell, J., 402 (Y. & S.)
King, P. Z., 73 (Misc.)
King, W. V., Roth, L., Toffaleti, J. & Middlekauff, W. W. (150) (Misc.)
Kirby, H., 290 (Am.)
Kirk, R., 121 (Y.F.)
——, with Stephenson, 316 (Misc.)
Kirkaldy-Willis, W. H., 217 (Am.)
Kirsh, D. & Diaz-Rivera, R. S., 218 (Am.)
Kirwan, E. O'G., Sen, K. & Bose, N., 511 (Oph.)
Kirzon, M. I., 261 (Mal.)
Kisner, P. & Lisansky, E. T., 751 (Fev.)
Kitchell, J. R., with Park & Shepherd, 493 (Lep.)
Kitchen, S. F. & Putnam, P., 5 *bis* (Mal.)
Kligler, I. J. & Olejnik, E., 199 (Typh.)
Klint, H., with Reading, 30, 203 (Fev.)
Kneedler, W. H., 450 (Mal.)
Knights, H. T., 957 (Hel.)
Knigin, T. D., with Bruce, Yolles & Graham, Jr., 93 (Mal.)
Knipe, F. W., with Russell & Sitapathy, 456 (Mal.)
Knowles, R. & Basu, B. C., 531 (Mal.)
Knutson, with Murray, 1007 (Mal.)
Kohls, G. M., with Cooley, (882) (Misc.)
——, with Parker, 277 (Fev.)
Kohlschütter, E., Zipf, H. F. & Triller, G., 454 (Mal.)
Kolodeznaja, Z. E., with Shukov-Vereshnikov, Zaletaev & Ibrazimova, 741 (Typh.)

Kondi, A., with Foy, 603 (Haem.)
——, with —— & Gluckman, 651 (Bl.)
Konj, J. S. & Yakovleva, O. I., 922 (Mal.)
Koprowski, H. & Lennette, E. H., 1030 *bis* (Y.F.)
Kornblueth, W., with Braun & Czertok, 186 (Mal.)
Kornetov, N. I. & Mirzoyan, N. A., 272 (Leish.)
Kossobudzki, S. L., with Fox & da Cunha, 123 (Y.F.)
Kouri, P. & Angulo, J. J., 1052 (Hel.)
Krasikova, V. I., with Jukov, 917 (Mal.)
Krause, G. R. & Crilly, J. A., 139 (Hel.)
Kremser, H., with Hesse, 1024 (Fev.)
Krontovskaya, M. K., 474 (Fev.)
Kubes, V. & Gallia, F., 905 (Rab.)
Kudicke, R., 660 (Typh.)
Kuhlmann, F. & Heinrich, K., 932 (Typh.)
Kumm, H. W. & Crawford, P. J., 123 (Y.F.)
—— & Zúniga, H., 643 (Mal.)
Kümmerling, K., 277 (Fev.)
Kuna, S., with Scudi & Jelinek, 728 (Mal.)
Kustner, H., with Kern, 644 (Mal.)
Kyles, W. B., with Agnew, 837 (Typh.)

L.

Ladell, W. S. S., 153, 782 (Misc.)
Laemmert, H. W., Jr., 747 (Y.F.)
—— & Moussatché, H., 123 (Y.F.)
Lahiri, K. D., 602 (Diet.)
Lalinde Del Rio, E., 514 (Misc.)
Lamy, L., 1056
——, with Deschiens, (1056) (Hel.)
Lancaster, H. O., with Lowe, 950, 951 (Hel.)
Landor, J. V., 71 (Der.)
Lane, J. & Cerqueira, N. L., 155 (B.R.)
——, with Galvão & Unti, (917) (Mal.)
Laouilheau, R., 999 (Mal.)
Larghero Ybarz, P. & Purriel, P. (139) (Hel.)
Larsen, K. & Lebel, H., 555 (Typh.)
Larsh, J. E., Jr., 764, 765 (Hel.)
Laskey, M., with Rothman, 218 (Am.)
Lasmar, J. E., with Martins, Versiani, Tupinamba, Sobrinho, Torres & Teixeira, 191 (Tryp.)
Lastres, J. B. & Anglas Quintana, P., 562 (Bart.)
Laurent, C., Barge, P., Berge, C., Audoye, H. & Fauconnier, J., 760 (Hel.)
Lavier, G., 368 (Tryp.)
Lavoipierre, M., with de Meillon, 679 (Hel.)
Layne, J. A., with Schwartz, Marvin & Watson, 503 (Diet.)
—— & Watson, C. J., 503 (Diet.)
——, with ——, 503 (Diet.)
Leake, W. H., with Simpson, McMahon, Gudex & Rueckert, 359 (Mal.)
Lebel, H., with Larsen, 555 (Typh.)
Lee, D. H. K., 416 (Diet.)
Leech, R. B., with de Meillon, 301 (Hel.)
Legant, O., with Zeligs & Webster, 752 (Fev.)
Legwen, W. A., (650) (Mal.)
Leishman, A. W. D. & Kelsall, A. R., 1062 (Misc.)
Leite, S. (965) (Der.)
Lendrum, A. C., 822 (Mal.)
Lenert, L. G., with Goodwin, Jr., 183 (Mal.)
Lennette, E. H., with Koprowski, 1030 *bis* (Y.F.)
—— & Perlowagora, A., 211 (Y.F.)
León, A. P. & Apodaca, F., 21, 379 (Typh.)
León, L. A., with Mazzotti, 192 (Tryp.)
Leon, R. C., with Basombrio, Mom & Noussitou, 404 (Lep.)
Lépine, P., Levaditi, J. C., Grabar, P. & Giuntini, J., 913 (Rab.)
Leprosy in India, 492, 1042 (Lep.)
Le Riche, H., 417 (Diet.)
——, with Kark, 775 (Misc.)
Leroy, G. & Linhard, J. (604) (Haem.)
LeRoy, G. V. & Lindberg, H. A., 1030 (Fev.)
Lesh, J. I. & Cannon, D. A., 134 (R.F.)
Levaditi, J. C., with Lépine, Grabar & Giuntini, 913 (Rab.)
Levenson, E. D., Fastorskaya, E. I., Khovanskaya, A. I. & Dukhanina, N. N., 10 (Mal.)
Lever, R. J. A. W., 150, 881 (Misc.)
Levin, I. L., 411 (Hel.)
Levin, N. B., with Carney, 730 (Mal.)
Levinson, S. O., Milzer, A., Shaughnessy, H. J., Neal, J. L. & Oppenheimer, F., 914 (Rab.)
Lewis, D. J., 213, 278 (Y.F.)
Li, Y. & Chien, Y. C., 56 (Hel.)
Lieberthal, E. P., 231 (Der.)
Liebesman, A., with Hitzig, 943 (R.F.)
Liebmann, H., with Herrlich, 782 (Misc.)
Liegeois, P., 1025 (Y.F.)
Lightner, G. H. & Patterson, W. B., 601 (Hel.)
Lillie, R. D., 74 *bis*, 876 (Misc.)
——, with Wright, 99 (Mal.)
Lima, S., with Soper, Wilson & Antunes, 699 (B.R.)
Lindberg, H. A., with Leroy, 1030 (Fev.)
Lindberg, K., 637 (Mal.)
Linhard, J., 14 *bis* (Tryp.)
——, with Leroy, (604) (Haem.)
Lipscomb, F. E. & Gibson, M. O. J., 550 (Leish.)
Lipshutz, J., with Ahlm, 745 (Fev.)
Lira, P. H., with Davis & Juvera, 744 (Typh.)
Lisansky, E. T., with Kisner, 751 (Fev.)
Livchitz, J. L., with Mochkovski, Diomina, Nossina, Pavlova, Pels & Roubtzova, 565 (Fev.)
Livesay, H. R. & Pollard, M., 209 (Fev.)
Livingood, C. S., Rogers, A. M. & Fitz-Hugh, T., Jr., 230 (Der.)
Livshitz, J. M., 566 (Fev.)
Lleras Restrepo, F., with Ignacio Chala, 587 (Lep.)
Loewenthal, L. J. A., 305 (Hel.)
Longinov, A. N., 140 (Hel.)
Lopez Soriano, P., 118 (Typh.)
Lörincz, F. & Jurány, E., 219 (Am.)
Loughlin, E. H., Bennett, R. H., Santora, E. & Mattucci, S., 186 (Mal.)
Lourie, E. M. & Collier, H. O. J., 293 (R.F.), 370 (Tryp.)
Louw, A. H., 839 (Typh.)
Lovera, I., 855 (Y. & S.)
——, with Vegas, Miguel Itriago & Medina, 856 (Y. & S.)

Loving, W. L., 580 (Lep.)
Low, G. C., 609 (Misc.)
Lowe, J., 825, 997 (Mal.)
Lowe, T. E., 969 (Misc.)
—— & Lancaster, H. O., 950, 951 (Hel.)
Lowman, E. W., 600 (Hel.)
Lowry, J. V., with Ashburn, (602) (Diet.)
Lozano Morales, A., 195 (Leish.)
Lozner, E. L. & Newhouser, L. R., 91 (Mal.)
Lübinsky, G. A., 573 (Am.)
——, with Nesterwodskaja, 102 (Mal.)
Luckhoff, C. A., 502 (Diet.)
Luis Carri, E., 737 (Leish.)
Lumley, G. F., 242 (B.R.)
Lummerzheim, H., 380 (Typh.)
Lumsden, W. H. R., (696) (Misc.)
Luttermoser, G. W., 592 (Hel.)
—— & Pifano, C. F., 590 (Hel.)
Lyle, T. K., Macrae, T. F. & Gardiner, P. A., 688 (Diet.)
Lyons, H. A., 855 (Y. & S.)

M.

Macaden, C. J. A., with Young, 724 (Mal.)
McAllister, J., with Augustine & Weinman, 759 (R.F.)
McArdle, B., 782 (Misc.)
McCarthy, D. D. & Brent, R. H., 214 (Fev.)
McCarthy, L., 312 (Der.)
Macchiavello, A., (39) (Pl.), 385 (Typh.)
McComas, G. & Martin, N. H., 466 (Tryp.)
McConn, C. F., 111 (Typh.)
McCoord, A. B., with Katsampes & Phillips, 852 (Am.)
McCord, C. P., 656 (Leish.)
McCoy, O. R., 612 (Misc.)
McCreary, J. F., with Tisdall & Pearce, 63 (Diet.)
Macdonald, G., 606 (Misc.)
McDowell, M. M., with Woodland & Richards, 34 (Fev.)
MacFarlane, W. V., 947 (Hel.)
McGill, A. F., 77 (Misc.)
McGovran, E. R. & Fales, J. H., 617 (Misc.)
MacGowan, 106 (Tryp.)
MacGregor, G. A., 949 (Hel.)
McGregor, T. & Eade, R. B., (617) (Misc.)
McGuire, C., 539 (Mal.)
Machado, L. M., 43 (Am.)
Machado González, A., 675 (R.F.)
Macht, D. I., 227 (Vms.)
MacInnes, D. G., with Glasgow, 6 (Mal.)
McKibbin, J. M., with Hegsted & Stare, 649 *bis* (Mal.)
McMahon, A., with Simpson, Leake, Gudex & Rueckert, 359 (Mal.)
McMahon, M. C. 753 (Pl.)
—— with Wayson 848 (Pl.)
McNamara K. N., 140 (Hel.)
MacNaught, W. W. & Murray-Lyon, R. M., 148 (Misc.)
MacQuillan, C. J., 316 (Misc.)
Macrae, T. F., with Lyle & Gardiner, 688 (Diet.)
Maegraith, B. G., 604 (Vms.)
Magalhães, O., 31 (Typh.), 843 (Fev.)
Magalhães & Guimarães, R., 228 (Vms.)
—— & Rocha, A., 31 (Typh.)
Magnoni, P., with Claveaux, Salveraglio & Canzani, 501 (Hel.)
Magnuson, H. J., with Eagle & Musselman, 942 (R.F.)
Maier, J. & Coggeshall, L. T., 826 (Mal.)
Maillard, E. R. & Hazen, E. L., 474 (Fev.)
Makhlina, R. M., 409 (Hel.)
Mandoul, H. & Mandoul, R., 311 (Der.)
Mann, H. D., with Carter & Smith, (617) (Misc.)
Mann, W. N., 100 (Mal.)
Manouélian, Y., 173 (Rab.)
Manson-Bahr, P., 80 (B.R.), 606, 609 (Misc.), 1003 (Mal.)
Manting, G., with Moore, 420 (Der.)
Manwell, R. D., 268 (Mal.)
Manwell, R. E. & Edgett, R., 189 (Mal.)
Manzullo, A., with Sordelli, Riessel & Ferrari, 388, 390 *ter*, 391 (Typh.)
Maplestone, P. A., 951 (Hel.)
Marangoni, B. A., with Palatucci, 1022 (Fev.)
Mareschal, P., with Sparrow, 276 (Typh.)
Mariano, J., 221, 1044 (Lep.)
Marill, F. G., 55 (Hel.)
Markovich, A. V., with Voroninaa, 1022 (Typh.)
Marques, A., 487 (R.F.)
Martin, J. T., (188) (Mal.)
Martin, N. H., with Findlay & Mitchell, 1028 (Y.F.)
——, with McComas, 466 (Tryp.)
Martin, R., with Parrot, (1012) *bis* (Leish.)
Martin, W. P. & Silber, B., 868 (Der.)
Martindale, 243, 244 (B.R.)
Martínez Báez, M., with Perrin, 233 (Der.)
Martins, A. V., Versiani, V., Tupinamba, A. A., Sobrinho, A. T., Torres, A., Lasmar, J. E. & Teixeira, A. A., 191 (Tryp.)
Martorelli, J., with Quetglas, 654 (Tryp.)
Marvin, J. F., with Schwartz, Layne & Watson, 503 (Diet.)
Matilla, V. & Aparicio, J., 530 (Mal.)
Mattucci, S., with Loughlin, Bennett & Santora, 186 (Mal.)
Maurer, F. D., with Jones, 937 (Y.F.)
Mauric, with Grosperrin, 78 (Misc.)
May, K. H., with Otto, 663 (Typh.)
May, M. Y., 44 (Am.)
Mayer, J. H. & Finlayson, M. H., 694 (Misc.)
Mayer, M., 562 (Bart.)
——, with Pifano, 589, 591 (Hel.)
Mayer, R. A., with Price, 930 (Leish.)
Mayfield, M. F., 697 (Misc.)
Mayoral, A., with Faget, 583 (Lep.)
Mazza, S., 376, 549 (Tryp.)
——, with Basso, G., Basso, R., Jórg, M. E. & Miyara, S., 16 (Tryp.)
—— & Chacon, R. V., 108 (Tryp.)
—— & Jauregui, R. B., 375 (Tryp.)
—— & Jörg, M. E., (830) (Tryp.)
—— & Oribe, H. R., 375 (Tryp.)
Mazzeo, M. & Sementini, A., 352 (Mal.)
Mazzotti, L., 134 (R.F.)
—— & León, L. A., 192 (Tryp.)
—— & Osorio, M. T., 16 (Tryp.), 957 (Hel.)
—— & Pastrana, A., 958 (Hel.)
——, Quintanar, E. & Prida, M., (306) (Hel.)

Mazzotti & Varela, G., 28 (Typh.)
——, with ——, 392 (Typh.)
Medical Journal of Australia, 934 (Typh.)
Medical Research Council, 700 *bis* (B.R.)
Medina, R., with Vegas, Lovera & Miguel Itriago, 856 (Y. & S.)
Megaw, J. W. D., 871 (Misc.)
——, with Rogers, 1072 (B.R.)
de Meillon, B., 182 (Mal.), 617 (Misc.)
——, with Gear & Davis, 739 (Typh.)
—— & Lavoipierre, M., 679 (Hel.)
—— & Leech, R. B., 301 (Hel.)
Melcher, L. R., 225 (Hel.)
Meleney, H. E., 612 (Misc.)
——, with Most, 536 (Mal.)
——, with Snyder, 216 (Am.)
Melnikova, Z. A., (448) (Mal.)
de Melo, V. H., (915) *bis* (Mal.)
Melville, A. R., with Wilson, D. B., 263 (Mal.)
Mendes de Castro, B., Giorgi, D. & Kieffer, J. C., 522 (B.R.)
Mengoni, H. R., with Steinberg, 295 (Lep.)
Menon, T. B., Ramamurti, B. & Sundarasiva Rao, D., 770 (Hel.)
Mera, B., 420 (Haem.)
Mercau, R., with Schujman, 51 (Lep.)
Merrill, E. D., 508 (Der.)
Messih, G. A., with Kamal, 380, 382 (Typh.)
Metcalf, R. L., with Patton, 546 (Mal.)
Metzner, S., 480 (Pl.)
Meunier, M., with Giroud, P. & Giroud, M. L., 738 (Typh.)
Meyer, H., 468 (Tryp.)
Meyer, K. F., Holdenried, R., Burroughs, A. L. & Jawetz, E., 398 (Pl.)
——, with Jawetz, 400, 753, 847, 1034, 1035 (Pl.)
——, with Sawyer, Eaton, Bauer, Putnam & Schwentker, 1026 (Y.F.)
Meyer, L. F., 41 (Dys.)
Meyer, P. F., with Don, 358 (Mal.)
Meyer, R., 115 *bis* (Typh.), 473 (Typh.)
Michael, P., 956 (Hel.)
Mickelsen, O., with Taylor, Henschel & Keys, 883 (Misc.)
Middlekauff, W. W. & Carpenter, S. J., (781) (Misc.)
——, with King, Roth & Toffaleti, (150) (Misc.)
Miguel Itriago, P., with Vegas, Lovera & Medina, 856 (Y. & S.)
Miller, E., with Stein, 426, 970 (Misc.)
Miller, J. J., Jr. & Wilbur, D. L., 497 (Hel.)
Miller, M. M., Silverman, J. J. & Powell, V. E., 786 (Misc.)
Milmore, B. K., 399 (Pl.)
Milzer, A., with Levinson, Milzer, Shaughnessy, Neal & Oppenheimer, 914 (Rab.)
Minckler, D., 861 (Hel.)
Ministry of Health, Dept. of Health for Scotland, (884) (Misc.)
Minning, W., 4 (Mal.)
——, with Zumpt, 4 (Mal.)
Mirzoyan, N. A., with Kornetov, 272 (Leish.)
Mitchell, J. B., with Findlay & Martin, 1028 (Y.F.)
Mitchell-Heggs, G. B., with Baker & Archer, 29 (Typh.)
Mitra, K., 63 (Diet.)
Miyara, S., with Mazza, Basso, G., Basso, R. & Jorg, 16 (Tryp.)
Mochkovski, C., 564 (Fev.)
——, Diomina, N. A., Nossina, V. D., Pavlova, E. A., Livchitz, J. L., Pels, H. J. & Roubtzova, V. P., 565 (Fev.)
Moeschlin, S., 294 (R.F.)
Mohr, J. L., with Mumford, 73, 965 (Misc.)
Mohsin Ali Abbasi, M., 910 (Rab.)
Mom, A. M., 582 (Lep.)
—— & Basombrio, G., 1049 (Lep.)
——, with ——, 1050 (Lep.)
——, with ——, Noussitou & Leon, 404 (Lep.)
Monchadsky, A. S., Blagoveschensky, D. I., Bregetova, N. G. & Ukhova, A. N., 1069 (Misc.)
Money, T. D. F., 587 (Lep.)
Montezin, G., with Funke & Bovet, 734 (Tryp.)
Monthly Bull. Ministry of Health & Emergency Pub. Health Lab. Service, 869 (Misc.)
Moore, M., Cooper, Z. K. & Weiss, R. S., 68 (Der.)
—— & Jorstad, L. H., 691 (Der.)
—— & Manting, G., 420 (Der.)
Moragues, V. & Pinkerton, H., 557, 558 (Typh.)
——, —— & Greiff, D., 839 (Typh.)
Morenas, L., 677 (Hel.)
Morgan, A. A. & Squires, B. T., 147 (Misc.)
Morishita, K., 395 (Fev.)
Mornas, P., 198 (Typh.)
Morse, E. M. & Seaton, S. P., 133 (Am.)
Morton, T. C., 606, 782 (Misc.)
Most, H. & Meleney, H. E., 536 (Mal.)
Motta, O. C., with Neves, 321 (Misc.)
Mourão, B. M., 962 (Der.)
Moussatché, H., with Laemmert, Jr., 123 (Y.F.)
Mozzotti, L. & Chavira, C., 861 (Hel.)
Mrugowsky, J., 202 (Typh.), 968 (Misc.)
Mudrow, L. & Bock, M., 1040 (R.F.)
——, with Reichenow, 190 (Mal.)
Muggleton, W. J., 80 (B.R.)
Mühlens, 934 (Typh.)
Muir, E., 52 (Lep.)
Mukerjee, S., with Roy, 103 (Mal.)
Mukerji, A. K. & Bhaduri, N. V., 595 (Hel.)
Mukerji, B., Ghosh, B. K. & Siddons, L. B., 9 (Mal.)
Mumford, E. P. & Mohr, J. L., 73, 965 (Misc.)
Munk, J., 484 (Am.)
Muraz, G., 734 (Tryp.)
Murdock, J. R., with Wright, 1054 (Hel.)
Murphy, F. D., with Polley, (686) (Hel.)
Murray, W. C. & Knutson, H., 1007 (Mal.)
Murray-Lyon, R. M., 609 (Misc.)
——, with Howie, 185 (Mal.)
——, with MacNaught, 148 (Misc.)
Muskatblit, E., with Tulipan, (70) (Der.)
Musselman, D., with Eagle & Magnuson, 942 (R.F.)
Muwazi, E. M. K. & Trowell, H. C., 615 (Misc.)

N.

Nachmansohn, D., with Waelsch, 649 (Mal.)
Nachtigal, D., with Bernkopf, 170 (Rab.)
Naidu, V. R., with Topping & Heilig, 29 (Typh.)
Nájera, L., 668 (Fev.)
Nance, F. D., 563 (Fev.)
Nancekievill, L., with Rumball & Parsons-Smith, 95 (Mal.)
Napier, L. E., 1054 (Hel.)
—— & Sen Gupta, P. C., 18 (Leish.), 143 (Haem.)
Nash, T. A. M., 653 *bis* (Tryp.)
Nataf, R., with Burnet, Cabasso & Cuénod, 1060 (Oph.)
National Research Council, 222 (Hel.)
Nattan-Larrier, L., Ronchèse, A. D. & Steeg, L., 931 (Leish.)
Nauss, R. W., 978 (B.R.)
Neal, J. L., with Levinson, Milzer, Shaughnessy & Oppenheimer, 914 (Rab.)
Neghme, A. & Ossandon, M., 300 (Hel.)
Negroni, P., with Baliña, Herrera & Bosq, 421 (Der.)
Nehaul, B. B. G., 512 (Misc.)
Nelson-Jones, A., 537 (Mal.)
Nesterwodskaja, K. M. & Lubinski, G. A., 102 (Mal.)
Nettel, R., 305 (Hel.)
Neubauer, D., with Anigstein, Bader & Young, 665 *bis* (Fev.)
Neubürger, K., with Jaffé, 1057 (Diet.)
Neujean, G., 1014 (Typh.)
Neumann, H., 954 (Hel.)
Neves, J. A. & Motta, O. C., 321 (Misc.)
—— & Sobrinho, O. P., 321 (Misc.)
Newbold, C. E., 11 (Mal.)
——, with Cochrane, 102 (Mal.)
Newhouser, L. R., with Lozner, 91 (Mal.)
Newman, H. R., (223) (Hel.)
Newton, B. L., with Kalz, 488 (Y. & S.)
Nicholls, L., 686 (Diet.)
Nicol, R., 62 (Diet.)
Nicol, W. D. & Shute, P. G., 71 (Misc.)
Nicole, J. E., (188) (Mal.)
Niño, F., with Ivanissevich, Taiana & Rosito, 284 (Am.)
Nissle, A., (929) (Tryp.)
Noble, G. A., 1070 (Misc.)
Nogueira Rivero, P., 221 (Hel.)
Nossina, V. D., with Mochkovski, Diomina, Pavlova, Livchitz, Pels & Roubtzova, 565 (Fev.)
Noth, P. H. & Hirshfeld, J. W., 757 (Am.)
Notley, F. B., with Wilson, 90 (Mal.)
Noussitou, F., with Basombrio, Mom & Leon, 404 (Lep.)
Nyka, W., 835 (Typh.)

O.

Obertreis, E., with Killian, 662 (Typh.)
O'Daly, J. A., 605 (Der.)
O'Donnell, R., with Beck & Bodily, 1020 (Typh.)
Oesterlin, M., with Bock, 545 (Mal.)
Offenkrantz, F. M., with Park, 471 (Typh.)
Oganesov, L. A., Sadoyan, V. S., Istamanian, L. S., Egnarian, V. I. & Karapetian, R. N., 535 (Mal.)
Ogborn, R. S., with Raper & Wilson, 647, (Mal.)
——, —— & Wright, 1000 (Mal.)
Oldham, F. K. & Kelsey, F. E., 260 (Mal.)
——, with ——, 260 (Mal.)
——; with —— & Geiling, 11 (Mal.)
——, with ——, —— & Dearborn, 923 (Mal.)
Oldham, J. N. & White, E. G., 769 (Hel.)
Olejnik, E., with Kligler, 199 (Typh.)
de Oliveira, J. C., 167 (Rab.)
Oliver, J. C., with Stuart-Harris & Rettie, 111 (Fev.)
Oliver-González, J., 223 (Hel.)
——, with Wright, 225 (Hel.)
Olivier, L. J. G., (650) (Mal.)
Olsen, Y. P., with Taliaferro, 15 (Tryp.)
Olson, C., with Glazier, 1070 (Misc.)
Olzscha, R., 256 (Mal.)
Oppenheimer, F., with Levinson, Milzer, Shaughnessy & Neal, 914 (Rab.)
Ordman, D., 853, 1037 (R.F.)
Oribe, H. R., with Mazza, 375 (Tryp.)
Orsini, O., 1068 (Misc.)
Osburn, H. S., 484 (Am.)
——, with Gelfand, 137 (Hel.)
Osorio, M. T., with Mazzotti, 16 (Tryp.), 957 (Hel.)
Osorno-Mesa, with Bugher, Boshell-Manrique & Roca-Garcia, 475 (Y.F.)
Ossandon, M., with Neghme, 300 (Hel.)
Ott, W. H., with Seeler & Gundel, 651 (Mal.)
Otto, R. & May, K. H., 663 (Typh.)
Ottolina, C. & Atencio, M. H., 945 (Hel.)
Oviedo Bustos, J. M., 237 (Misc.)

P.

Packchanian, A., 107, 270 (Tryp.), 823 (Mal.)
——, with Ashburn, 488 (R.F.)
Page, J. E., with Goodwin, 20 (Leish.), 222 (Hel.)
Page, N., with Futcher & Consolazio, 973 (Misc.)
Paine, R. W., 328 (B.R.)
Palatucci, O. A. & Marangoni, B. A., 1022 (Fev.)
Palestine, 939 (Pl.)
Palmer, H. A., (976) (Misc.)
Panja, D. & Ghosh, L. M., 614 (Misc.)
Panja, G. & Ghosh, S. K., 40 (Chl.)
Parade, G. W., 820 (Mal.)
Pardo, L., with Pesce, 110 (Leish.)
Pardo-Castello, V. & Tiant, F. R., 135, 585 (Lep.)
Park, F. R., Kitchell, J. R. & Shepherd, S. G., 493 (Lep.)
Park, J. H. & Offenkrantz, F. M., 471 (Typh.)
Parker, R. R. & Kohls, G. M., 277 (Fev.)
Parkin, E. A. & Green, A. A., (187) (Mal.), 880 (Misc.)
Parkins, P. V., with Ratcliffe, 941 (Am.)
Parr, H. C. M., (320) (Misc.)
Parran, T., 612 (Misc.)

Parrot, L., 696 (Misc.), (1012) *bis* (Leish.)
——, Catanei, A., Collignon, E. & Ambialet, R., 539 (Mal.)
—— & Clastrier, J., (1012) (Leish.)
—— & Gougis, R., 736, (1012) (Leish.)
—— & Martin, R., (1012) *bis* (Leish.)
Parsons-Smith, B. G., 693 (Misc.)
——, with Rumball & Nancekievill, 95 (Mal.)
Paskind, L., with Annegers, Snapp, Ivy & Atkinson, 98 (Mal.)
Passmore, R., with Shone, 184 (Mal.)
Pastrana, A., with Mazzotti, 958 (Hel.)
Patel, J. C., 118 (Typh.)
Patel, N. D., 387 (Typh.)
Patrick, F. L. L., 781 (Misc.)
Patterson, W. B., with Lightner, 601 (Hel.)
Patton, R. L. & Metcalf, R. L., 546 (Mal.)
Patzer, G., with Wohlrab, 741 (Typh.)
Paul, J. R., with Sabin & Philip, 1031 (Fev.)
Pavlova, E. A., with Mochkovski, Diomina, Nossina, Livchitz, Pels & Roubtzova, 565 (Fev.)
Pavlovsky, E. N., 129 (Fev.), 513 (Misc.), 673 (R.F.)
Pearce, H., with Tisdall & McCreary, 63 (Diet.)
Peddie, J. J. G., with Cottrell & Stronach, 1033 (Fev.)
Peel, E., with van Hoof & Henrard, 928 (Tryp.)
Pei, Y. S., with Yao & Wu, 999 (Mal.)
Pels, H. J., with Mochkovski, Diomina, Nossina, Pavlova, Livchitz & Roubtzova, 565 (Fev.)
Pemberton, C. E., 430, 616 (Misc.)
Peña Chavarria, A., Sáenz Herrera, C. & Cordero, E., 551 (Leish.)
Peña-Yánez, A., 192 (Leish.)
Penfold, J. B., 387 (Typh.)
Penna, H. A., with Fox, 125 (Y.F.)
Pérez Gallardo, F., with Clavero, 24 (Typh.)
Pérez Hurtado, F., with Valdés Dapena, 1052 (Hel.)
Perlowagora, A., with Lennette, 211 (Y.F.)
van Pernis, P. A., Benson, M. E. & Holinger, P. H., 70 (Der.)
Perrin, T. G. & Martinez Báez, M., 233 (Der.)
Persia and Iraq Force, 74 (Misc.)
Perves, M., 354 (Mal.)
Peryassú, A. G., 940 (Pl.)
Pesce, H. & Pardo, L., 110 (Leish.)
Pessôa, S. B., 1061 (Oph.)
—— & Barreto, M. P., 189 (Mal.)
Pessôa, S. S. B. & Barreto, M. P., 544, 732 (Mal.)
Petazzi, A., 353 (Mal.)
Peters, F., with Kathe, (142) (Hel.)
Peters, M., with Colvin & Gore, 774 (Der.)
Peterson, O. L., 935 (Typh.)
Peyrallo, R., with Chifflet, (139) (Hel.)
Pfanner, W., 671 (Am.)
Pfeffer, G. & Gauwerky, F., 274 (Typh.)
Phatak, N. M., with David & Zener, 672 (Am.)
Philip, C. B., with Plotz, Woodward, Bennett & Evans, 28 (Typh.)
——, with Sabin & Paul, 1031 (Fev.)
Phillips, J. W., 910 (Rab.)
Phillips, W. A., with Katsampes & McCoord, 852 (Am.)
Pick, E. P. & Hunter, J., 924 (Mal.)
Pierce, J. S., with Ballard, (729) (Mal.)
Pierson, P., with Holman, 762 (Hel.)
Pifano, C. F., 576 (R.F.)
——, with Lüttermoser, 590 (Hel.)
—— & Mayer, M., 589 (Hel.)
——, with ——, 591 (Hel.)
Pimentel Imbert, M. F., 232 (Der.)
Pinkerton, H., with Greiff, 840 (Typh.)
——, with Moragues, 557, 558 (Typh.)
——, with —— & Greiff, 839 (Typh.)
Pinto, M. R., 27 (Typh.)
Piper, B. C., with Denhoff, 918 (Mal.)
Pirot, R. & Bourgain, M., 744 (Typh.)
Pirzada, M. A., 851 (Am.)
Pitt, L. A., 51 (Lep.)
——, with Garzon, 135 (Lep.)
Pitta Simões, J. M. & Hill, R. B., 496 (Hel.)
Pitts, G. C., with Johnson & Consolazio, 974 (Misc.)
Piza, J. de T., 208 (Fev.)
Placeres, M., (363) (Mal.)
Platonov, N. V., 636 (Mal.)
—— & Tarabukhin, I. A., 541 (Mal.)
Plaza Izquierdo, L., 677 (Hel.)
Plotz, H., Reagan, R. L. & Wertman, K., 842 (Fev.)
——, with Wertman, 659 (Typh.)
——, —— & Reagan, R. L., 1023 (Fev.)
——, Woodward, T. E., Philip, C. B., Bennett, B. L. & Evans, K. L., 28 (Typh.)
Plum, C. M., 423 (Misc.)
Pochard, P., (324) (Misc.)
Podyapolskaya, V. P., 301 (Hel.)
Pogge, R. C., with Faget, Johansen, Dinan, Prejean & Eccles, 494 (Lep.)
Pollard, M., with Livesay, 209 (Fev.)
Polley, T. Z. & Murphy, F. D. (686) (Hel.)
Pollock, F. J., with Venables, (512) (Oph.)
Polumordvinov, A. D., 541, 823 (Mal.)
Porges, R., 482 (Pl.)
Portius, W., with Ernst, 559 (Fev.)
Portnoy, B., with Boyd, 569 (Dys.)
Potenza, L., 479 (Y.F.)
Poth, E. J., 570 (Dys.)
—— & Ross, C. A., 757 (Dys.)
Potter, C., with Tattersfield, (187) (Mal.)
Potter, L. 782 (Misc.)
Potter, W. B., 370 (Tryp.)
Powell, V. E., with Miller & Silverman, 786 (Misc.)
Prasad, B. N. & Chowdhury, N. K., 602 (Haem.)
Prather, R. M., Jr., with Redmond, 267 (Mal.)
Pratt, H. D., (356) (Mal.)
——, with Pritchard, 722 (Mal.)
Prejean, B. M., with Faget, Pogge, Johansen Dinan & Eccles, 494 (Lep.)
Prescott, F., with Clarke, 62 (Diet.)
Preston, P. G., 314 (Misc.)
Pretorius, T. P., with de Waal, (149) (Misc.)
Price, F. L. & Mayer, R. A., 930 (Leish.)
Prida, M., with Mazzotti & Quintanar, (306) (Hel.)
Prince, F. M., 37 (Pl.)
Prior, A. P., 58 (Hel.)
di Prisco, J., 946 (Hel.)
Pritchard, A. E. & Pratt, H. D., 722 (Mal.)

Prudhomme, R. O., 588 (Lep.)
Pshènichnov, A. W., 836 (Typh.)
Public Health Rep., Wash., 36 (Pl.), 844 (Fev.)
Puhlmann, H., 919 (Mal.)
Purandare, N. M., with Dhayagude, 533 (Mal.)
Purriel, P., with Larghero Ybarz, (139) (Hel.)
Putnam, P., with Kitchen, 5 *bis* (Mal.)
——, with Russell & Badri Nath Mohan, 104 (Mal.)
——, with Sawyer, Meyer, Eaton, Bauer & Schwentker, 1026 (Y.F.)
Puttaya, M., with Heilig, 428 (Misc.)

Q.

Quetglas, A. & Martorelli, J., 654 (Tryp.)
Quinland, W. S., with Turner, Bent, Holloway & Cuff, 148 (Misc.)
Quintanar, E., with Mazzotti & Prida, (306) (Hel.)

R.

Rachmilewitz, M., (758) (Am.)
Raettig, H., 275, 932 (Typh.)
Raevsky, G. E., 541 *ter* (Mal.)
Rajagopalan, G., with Cochrane, 491 (Lep.)
Rajindar Pal, 448, (641) (Mal.), 972 (Misc.)
Rakhmanova, P. I., 541 (Mal.)
Ramachandra Rao, T., with Viswanathan, 641 (Mal.)
Ramakrishna, V., with White, Adhikari & Roy, 720 (Mal.)
Ramamurti, B., with Menon & Sundarasiva Rao, 770 (Hel.)
Raman, T. K., 60 (Diet.)
Ramos, A. S., (917) (Mal.)
——, with Unti, (917) (Mal.)
Randall, R. & Cooper, H. K., 1038 (R.F.)
Rao, A. S., 1041 (Lep.)
Rao, M. A., with Sundaresan, (449) (Mal.)
Rao, S. S., 59 (Hel.)
Rao, V. G., with Hardikar, 319 (Misc.)
Rao, V. V., with White, 720 (Mal.)
Raper, A. B., Ogborn, R. S. & Wilson, D. B., 647 (Mal.)
——, with —— & Wright, 1000 (Mal.)
Rasberry, E. A., with Diaz-Rivera, 1036 (Am.)
Rashna, M. G., 447 (Mal.)
Ratcliffe, H. L., 857 (Hel.)
—— & Parkins, P. V., 941 (Am.)
Ravelo-Barré, M. & Thomen, L. F., 289 (Am.)
Raven, R. W., 537 (Mal.)
Ray, H. N., 1070 (Misc.)
Reading, B. & Klint, H., 30, 203 (Fev.)
Reagan, R. L., with Plotz & Wertman, 842, 1023 (Fev.)
Reardon, L. V., with von Brand, Rees & Jacobs, 133 (Am.)
Rebêlo, A. & de Carvalho Pereira, M., (237) (Misc.)
Rector, N. H., 650 (Mal.)
Redmond, W. B. & Prather, R. M., Jr., 267 (Mal.)
Reed, A. C., & Geiger, J. C., 886 (B.R.)
Rees, C. W., with von Brand, Jacobs & Reardon, 133 (Am.)
Regamey, R., 200 (Typh.)
Registrar-General, England and Wales, 700 (B.R.)
Reichenow, E. & Mudrow, L., 190 (Mal.)
Reimer, K., 120 (Fev.)
Remennikova, V. M., 817 (Mal.)
Remlinger, P. & Bailly, J., 904 (Rab.)
Reniger-Aresheva, M. L., 919 (Mal.)
Renn, C. E., 182 (Mal.)
Renoux, G., with Benhamou & Horrenberger, 25 (Typh.)
Republica Argentina, 638 (Mal.)
Rettie, G. K. C., with Stuart-Harris & Oliver, 111 (Fev.)
Revol, P., with Giraud, 109 (Leish.)
Rey, H., 110 (Leish.)
Reyniers, J. A., 81 (B.R.)
Ribbands, C. R., 925 (Mal.)
Richard, H., with Sergent, 291 (R.F.)
Richards, J. T., with Woodland & McDowell, 34 (Fev.)
Richardson, A. P., with Hewitt, 188 (Mal.)
Rico, with Calderon, Barca & Rico-Avello, 200 (Fev.)
Rico-Avello, with Calderon, Barca & Rico, 200 (Fev.)
Riessel, M., with Sordelli, Manzullo & Ferrari, 388, 390 *ter*, 391 (Typh.)
Rivas, C. I., (949) (Hel.)
Roberts, F. H. S., 641 (Mal.)
Roberts, H. R., with Ross, 256 (Mal.)
Roberts, T. L. & Daniels, W. B., 41 (Dys.)
Robertson, F., 106 (Bl.)
Robinson, G. G., 292, 674 (R.F.), 972 (Misc.)
Robinson, S., Turrell, E. S., Belding, H. S. & Horvath, S. M., 520 (Misc.)
Roca-Garcia, M., with Bugher, Boshell-Manrique & Osorno-Mesa, 475 (Y.F.)
Rocha, A., with Magalhães, 31 (Typh.)
Rockefeller Foundation, 242 (B.R.)
Rodeiro, M., with de Villafañe Lastra, 399 *bis* (Pl.)
——, with ——, Goobar & Videla, 400 (Pl.)
Rodger, L. M., 726 (Mal.)
Rodhain, J., 605 (Der.)
—— & Dellaert, R., 544 (Mal.)
Rodriguez de la Cruz, J. M., with Roig & Acuña, (1045) (Lep.)
Rodriguez, J. N., with Douil, Guinto & Bancroft, 48 (Lep.)
Rodriguez Navarro, M., 867 (Der.)
Rogan, J. M., 921 (Mal.)
Rogers, A. M., with Livingood & Fitz-Hugh, Jr., 230 (Der.)
Rogers, L., 53 (Lep.)
——, with D'Abreu, (139) (Hel.)
—— & Megaw, J. W. D., 1072 (B.R.)
Rohrschneider, W., 1060 (Oph.)
Roig, J. T., Acuña, J. & Rodriguez de la Cruz, J. M., (1045) (Lep.)
Rojas, M., with Hermosilla Diaz & Awad, (735) (Tryp.)
Rome, H. P. & Fogel, R. H., 304 (Hel.)
Romeiro, O. dos S., (550) (Tryp.)
Romero Jordan, O., with Viamonte Cuervo, Alonso Suarez, Castro Palomino & Grau Triana, (1045) (Lep.)
Romero Moliner, R., 516 (Misc.)
Romero Pérez, M., with Camacho Téllez, Diaz Angulo & Salazar, 203 (Typh.)

Romeu Cançado, J., 53 (Hel.)
Ronchèse, A. D., with Nattan-Larrier & Steeg, 931 (Leish.)
Rose, H. M., 60 (Hel.)
——, with Culbertson, 772 (Hel.)
——, with —— & Demarest, 770, 771 (Hel.)
Rosito, E., with Ivanissevich, Taiana & Niño, 284 (Am.)
Ross, C., with Davey, 587 (Lep.)
Ross, C. A., with Poth, 757 (Dys.)
Ross, E. S. & Roberts, H. R., 256 (Mal.)
Ross, H., with Faget, 1046 (Lep.)
Rotenburg, S. S., with Tarejev & Gontayeva, 257 (Mal.)
Roth, H., 59 (Hel.)
Roth, J., 551 (Leish.)
——, with Süsskind, 377 (Leish.)
Roth, L., with King, Toffaleti & Middlekauff, (150) (Misc.)
Rothman, M. M. & Laskey, M., 218 (Am.)
Roubaud, E. & Girard, G., 567 (Pl.)
—— & Grenier, P., 881 (Misc.)
Roubtzova, V. P., with Mochkovski, Diomina, Nossina, Pavlova, Livchitz & Pels, 565 (Fev.)
Rowley, H. F., with Rubenstein, 119 (Typh.)
Roy, A. N. & Mukerjee, S., 103 (Mal.)
Roy, B. B., with White, Adhikari & Ramakrishna, 720 (Mal.)
Roy, B. C. & Chatterjee, K. D., (1006) (Mal.)
Roy, D. N., 449 (Mal.)
—— & Ghosh, S. M., 972 (Misc.)
Roy, S. C., with Chakravarty, (426) (Misc.)
Roy, V. V., with White, 720 (Mal.)
Royal Naval Medical Bulletin, 510 (Oph.), 953 (Hel.)
Royo Montañés, M., 133 (Am.), 514 (Misc.)
Rozeboom, L. E., with Russell & Stone, 325 (B.R.)
Rubenstein, A. D. & Rowley, H. F., 119 (Typh.)
Rubin, S. S., 680 (Hel.)
Rubinstein, B. N., Sacharova, P. I. & Yakovleva, E. S., 659 (Typh.)
Rueckert, R. R., with Simpson, Leake, McMahon & Gudex, 359 (Mal.)
Rugiero, H. R., with Gandolfo, Steinberg, Del Ponte, Crivellari & Coria, 311 (Der.)
Ruiz Castañeda, M., 1018 (Typh.)
Ruiz Rodriguez, J. M., (857) (Hel.)
Ruiz Sánchez, F., 290 (Am.), 942 (R.F.)
Rumball, C. A., Parsons-Smith, B. G. & Nancekievill, E., 95 (Mal.)
Russell, J. C., with Boyd, 92 (Mal.)
Russell, P. F., 356 (Mal.)
——, Badri Nath Mohan & Putnam, P., 104 (Mal.)
——, Knipe, F. W. & Sitapathy, N. R., 456 (Mal.)
——, Rozeboom, L. E. & Stone, A., 325 (B.R.)

S.

Sá Antunes, W. S., (479) (Y.F.)
Sabin, A. B., Philip, C. B. & Paul, J. R., 1031 (Fev.)
Sabrosky, C. W., (917) (Mal.)
Sacharova, P. I., with Rubinstein & Yakovleva, 659 (Typh.)
Sachdeva, Y. V., 470 (Leish.)
Sachs, A., 782 (Misc.)
Sadoyan, V. S., with Oganesov, Istamanian, Egnarian & Karapetian, 535 (Mal.)
Sáenz Herrera, C., with Peña Chavarria & Cordero, 551 (Leish.)
Sáenz Vera, C., 481 (Pl.)
Sagatelova, I. S., with Kalandadze, 916 (Mal.)
Sagebiel, J. L., with Simpson, 360 (Mal.)
Sagel, W., 356 (Mal.)
Sakzshevsky, E. B., 644 (Mal.)
Salazar, C. S., 1045 (Lep.)
Salazar, G., with Camacho Téllez, Diaz Angulo & Romero Pérez, 203 (Typh.)
Salveraglio, F. J., with Claveaux, Magnoni & Canzani, 501 (Hel.)
Sampayo, R. R. L., 310 (Vms.)
Sanabria, A., 593 (Hel.)
Sancho Lobo, M., 555 (Typh.)
Sandground, J. H., 372 (Tryp.)
—— & Hamilton, C. R., 372 (Tryp.)
Sandstead, H. R., 864 (Diet.)
Santora, E., with Loughlin, Bennett & Mattucci, 186 (Mal.)
Santos, A. B., 1005 (Mal.)
Santra, I., 1042, 1043 *bis* (Lep.)
——, with Dharmendra, 1045 (Lep.)
Sapre, S. N., (1038) (R.F.)
Sarikian, S. J., 817 (Mal.)
Sarmento, A., 997 (Mal.)
Saunders, G. M., 49 (Lep.)
—— & Giffen, H. K., 50 (Lep.)
—— & Guinto, R. S., 49 (Lep.)
Sauter, H. B., 1020 (Typh.)
Sawitz, W. G. & Hammerstrom, R. J., 43 (Am.)
Sawyer, W. A., 612, 967 (Misc.), 787 (B.R.)
——, Meyer, K. F., Eaton, M. D., Bauer, J. H., Putnam, P. & Schwentker, F. F., 1026 (Y.F.)
Scadding, J. G., 755, 1036 (Dys.)
Scheng, T. D., 366 (Mal.)
Schiavi, A., 915 (Mal.)
Schilling, C., 723 (Mal.)
Schlesinger, C. J., with Eigenfeld, 496 (Hel.)
Schlumberger, H. G. & Service, A. C., 606 (Der.)
Schmidt, H., with Kikuth, 194 (Leish.)
Schneyer, L., with Johnson, 923 (Mal.)
Schorr, S., with Druckmann, 850 (Am.)
Schreiber, W., 686 (Hel.)
Schüffner, W. & Bohlander, H., 675 (R.F.)
Schujman, S. & Mercau, R., 51 (Lep.)
Schulten, H. & Broglie, 127 (Fev.)
Schwartz, S., Marvin, J. F., Layne, J. A. & Watson, C. J., 503 (Diet.)
Schwentker, F. F., with Sawyer, Meyer, Eaton, Bauer & Putnam, 1026 (Y.F.)
Schwetz, J., 90, 529 (Mal.)
Scott, H. H., 71, 234, 606, 609, 782 (Misc.)
Scott, J. G., 683 (Hel.), 688 (Diet.), 733 (Tryp.), 920 (Mal.)
Scudi, J. V. & Hamlin, M. T., 262, 263, 728 (Mal.)
—— & Jelinek, V. C., 452 (Mal.)
——, —— & Kuna, S., 728 (Mal.)
Seaton, S. P., with Morse, 133 (Am.)

Secret, E., 661 (Typh.)
Seeler, A. O., Ott, W. H. & Gundel, M. E., 651 (Mal.)
Seelig, S. F. & Hemming, J. R., 724 (Mal.)
Segal, A. E. & Zasosova, L. I., 386 (Typh.)
Seiferth, L. B., 933 (Typh.)
Sekar, C. C., 143 (Haem.)
Semenova, N. E., with Shikhobalova, 411 (Hel.)
Sementini, A., with Mazzeo, 352 (Mal.)
Sen, K., with Kirwan & Bose, 511 (Oph.)
Senekjie, H. A., 548 (Tryp.)
Sen Gupta, P. C., 17, 273, 736 *bis* (Leish.), 144 (Haem.)
——, with Das Gupta, 468 (Leish.)
——, with Napier, 18 (Leish.), 143 (Haem.)
Sen Gupta, S. K., 401 (Chl.)
Sergent, A. & Richard, H., 291 (R.F.)
Sergent, E., 65, 691, 773 (Vms.)
Sergent, Et., (65) (Vms.)
Serras Simões, T., 516 (Misc.)
Service, A. C., with Schlumberger, 606 (Der.)
Shakirzianova, M. S., (617) (Misc.)
Shah, R. L., (426) (Misc.)
Shannon, R. C., with Downs & Gillette, 445 (Mal.)
Shapiro, B. G., 665 (Typh.)
Shaughnessy, H. J., with Levinson, Milzer, Neal & Oppenheimer, 914 (Rab.)
—— & Zichis, J., 173 (Rab.)
Shaw, F. H., with Keogh, 365, 1003 (Mal.)
Shearer, W. S., 616 (Misc.)
Sheinker, K. P., 541, 817 (Mal.)
Shelley, H. M., 203 (Typh.)
Shellim, M. A., 833 (Leish.)
Shepherd, S. G., with Park & Kitchell, 493 (Lep.)
Sheppard, E. M., with Duguid, 857 (Hel.)
Shih Lu Chang, 670 (Am.)
Shikhobalova, N. P. & Semenova, N. E., 411 (Hel.)
Shi Lu Chang, 217 (Am.)
Shipitsina, N. K., 719 (Mal.)
Shircore, J. O., 778 (Misc.)
Shishliaeva-Matova, Z. S., 817, 819 (Mal.)
Shone, S. & Passmore, R., 184 (Mal.)
Shterngold, E. J., with Khodukin, 939 (Fev.)
Shukov-Vereshnikov, N. N., Kolodeznaja, Z. E., Zaletaev, S. G. & Ibrazimova, N. V., 741 (Typh.)
Shute, P. G., 255, 528, 1002 (Mal.)
——, with Nicol, 71 (Misc.)
Sicart, M., (696) (Misc.)
Siddons, L. B. & Bose, A. N., 826 (Mal.)
——, with Das Gupta, 11, 99, 545, 826 (Mal.)
——, with Mukerji & Ghosh, 9 (Mal.)
Sigurdsson, B., 169 (Rab.)
Silber, B., with Martin, 868 (Der.)
Silverman, J. J., with Miller & Powell, 786 (Misc.)
Simmons, J. S., 355 (Mal.)
Simpson, W. M., Leake, W. H., McMahon, A., Gudex, T. V. & Rueckert, R. R., 359 (Mal.)
—— & Sagebiel, J. L., 360 (Mal.)
Simpson, T., 778 (Misc.)
Singh, I. & Singh, I., 927 (Bl.)
Singh, J. & Bhattacharji, L. M., 822 (Mal.)
Sinton, J. A., 257 (Mal.)
Sippe, G. R., 689 (Haem.)
Sisk, W. N., 355 (Mal.)
Sitapathy, N. R., with Russell & Knipe, 456 (Mal.)
Sivalingam, V., 718 (Mal.)
Sizemore, P., 206 (Fev.)
Skuja, N., 121 (Fev.)
Smart, J., 328 (B.R.)
Smith, C. M., with Carter & Mann, (617) (Misc.)
Smith, D. C., 680 (Hel.)
Smith, H. H., Bevier, G. & Bugher, J. C., 210 (Y.F.)
——, with Bugher, 477 (Y.F.)
Smith, J., with Beckman, 267, 545 (Mal.)
Smithers, R. H. N., 229 (Vms.)
Smorodintzeff, A. A. & Fradkina, R. V., 1021 (Typh.)
Snapp, F. E., with Annegers, Paskind, Ivy & Atkinson, 98 (Mal.)
Snavely, J. R., with Turner, Grossman, Buchanan & Foster, 750 (Y.F.)
Sneddon, I. B., 259 (Mal.)
Snell, T. B., 76 (Misc.)
Snodgrass, R. E., 880 (Misc.)
Snow, J. S. & Harley, R. D. (774) (Der.)
Snyder, T. L. & Meleney, H. E., 216 (Am.)
Sobrinho, A. T., with Martins, Versiani, Tupinamba, Torres, Lasmar & Teixeira, 191 (Tryp.)
Sobrinho, O. P., with Neves, 321 (Misc.)
Sokhey, S. S. &Habbu, M. K., 129 *bis* (Pl.)
—— & Wagle, P. M., 37 (Pl.)
Solsona Conillera, J., 493 (Lep.)
van Someren, G. R. C., 257, (881) (Mal.)
Somers, R. B. U., 654 (Leish.)
Sommer, E., 680 (Hel.)
Sommerville, E. T. W., with Dios, 829 (Tryp.)
Soper, F. L. & Wilson, D. B., 324 (B.R.)
——, ——, Lima, S. & Antunes, W. S., 699 (B.R.)
Soper, H. W., 290 (Am.)
Sordelli, A., Manzullo, A., Riessel, M. & Ferrari, J., 388, 390 *ter*, 391 (Typh.)
Sorsby, A., 1060 (Oph.)
Sosa, H. & Vidal de Gambino, R., 389 *bis* (Typh.)
Soshnikova, M. N., with Khodukin & Kevorkova, 938 (Fev.)
Soto, M. C., 405, 406 (Lep.)
de Souza-Araujo, H. C., 220, (1044) (Lep.)
Spaet, G., 931 (Typh.)
Sparrow, H. & Mareschal, P., 276 (Typh.)
Spies, T. D., with Bean & Blankenhorn, 959 (Diet.)
——, with —— Vilter, 960, 1058 (Diet.)
Sporer, A., with Symmers, 1059 (Der.)
Springall, A. N., 645 (Mal.)
Squires, B. T., 147 (Misc.)
——, with Morgan, 147 (Misc.)
Stacey, R. S., 779 (Misc.)
Stannus, H. S., 606, 609 (Misc.), 862 (Diet.)
Stare, F. J., with Hegsted & McKibbin, 649 *bis* (Mal.)
Stavisky, J. D., 487 (R.F.)
Steeg, L., with Nattan-Larrier & Ronchèse, 931 (Leish.)
Steele, C. W., with Beattie, Herbert & Wechtel, 409 (Hel.)
Stein, G. J., 575 (R.F.)

Stein, H. B. & Miller, E., 426, 970 (Misc.)
Steinberg, I. R. & Crivellari, C. A., 912 (Rab.)
——, with Gandolfo, Rugiero, Del Ponte, Crivellari & Coria, 311 (Der.)
—— & Mengoni, H. R., 295 (Lep.)
Stephenson, R. W. & Kirk, R., 316 (Misc.)
Sternfeld, G., 551 (Leish.)
Steuer, W., 113 (Fev.)
Stewart, F. H., 937 (Fev.)
Stewart, M. A., (938) (Fev.), (957) (Hel.)
Stirk, E. M., (Mal.)
Stiven, D., with Jamieson & Brodie, 754 (Dys.)
v. Stockert, F. G., 201 (Typh.)
Stone, A., with Russell & Rozeboom, 325 (B.R.)
Stronach, R. D., with Cottrell & Peddie, 1033 (Fev.)
Stuart-Harris, C. H., Rettie, G. K. C. & Oliver, J. C., 111 (Fev.)
Stylianakis, V. G., 510 (Oph.)
Subbaraman, A. K. & Vedamanikkam, J. C., 457 (Mal.)
Suesskind, S., 971 (Misc.)
Sulkin, S. E. & Harford, C. G., 171 (Rab.)
Sullivan, T. D., 1011 (Tryp.)
Sundarasiva Rao, D., with Menon & Ramamurti, 770 (Hel.)
Sundaresan, B. & Rao, M. A., (449) (Mal.)
Süsskind, S. & Roth, J., 377 (Leish.)
Sutton, R. L., Jr., 430 (Misc.)
Swartz, H., 278 (Y.F.)
Sylla, A., 128 (Fev.)
Symmers, D. & Sporer, A., 1059 (Der.)
Symonds, J. W. C., 293 (R.F.)
Szidat, L., 57 (Hel.)

T.

Taiana, J. A., with Ivanissevich, Niño & Rosito, 284 (Am.)
Talbot, D. R., 97 (Mal.)
Taliaferro, W. H. & Olsen, Y. P., 15 (Tryp.)
Tanganyika Territory: Department of Tsetse Research, 460 (Tryp.)
Tarabukhin, I. A., with Platonov, 541 (Mal.)
Tareev, E. M., 981 (B.R.)
Tarejev, E. M., Gontayeva, A. A. & Rotenburg, S. S., 257 (Mal.)
Tattersfield, F. & Potter, C., (187) (Mal.)
Taylor, F. H., 81, 242, 698 (B.R.)
Taylor, H. L., Henschel, A. F. & Keys, A., 322 (Misc.)
——, with —— & —— 882 (Misc.)
——, ——, Mickelsen, O. & Keys, A., 883 (Misc.)
Tchernomoretz, J., with Adler, 103, 366, 458 (Mal.)
Teixeira, A. A., with Martins, Versiani, Tupinamba, Sobrinho, Torres & Lasmar, 191 (Tryp.)
Teriteanu, E., with Ciuca, Badenschi & Constantinescu, 645 (Mal.)
Theiler, G., 1024 (Fev.)
Thiagarajah, P. R., 409 (Hel.)
Thiersch, J. B., 1068 (Misc.)
Thomas, H. D., (213) (Y.F.)
Thomas, P. E., with Baranger, 103 (Mal.)
Thomen, L. F., with Ravelo-Barré, 289 (Am.)
Thompson, P. E. & Huff, C. G., 827 *bis* (Mal.)
Thompson, R. B., 273 (Leish.)
Thomson, E. L., 601 (Diet.)
Thonnard-Neumann, E., 362 (Mal.)
Thuringer, J. M., 774 (Der.)
Tiant, F. R., with Pardo-Castello, 135, 585 (Lep.)
Tisdall, F. F., McCreary, J. F. & Pearce, H., 63 (Diet.)
Tixier, R., with Chorine, Grabar & Crougue, 291 (R.F.)
Toffaleti, J., with King, Roth & Middlekauff, (150) (Misc.)
Tokarevich, K. N., 1017 (Typh.)
—— & Epstein, E. F., 1017 (Typh.)
Tom, N., 376 (Tryp.)
Tomb, J. W., 71 (Der.)
Tomé Bona, J. M., 1052 (Lep.)
Tommaso, P., 182 (Mal.)
Topping, N. H., 740 (Typh.)
——, Heilig, R. & Naidu, V. R., 29 (Typh.)
Torrealba, J. F., 830 (Tryp.)
Torres, A., with Martins, Versiani, Tupinamba, Sobrinho, Lasmar & Teixeira, 191 (Tryp.)
Torres Cañamares, F., (1069) (Misc.)
Torrey, J. C., 978 (B.R.)
Totterman, G., 858 (Hel.)
Townsend, S. L., 480 (Pl.)
Transactions of the Royal Society of Tropical Medicine and Hygiene, 71 (Misc.)
Treu, R., 758 (Am.)
Triller, G., with Kohlschütter & Zipf, 454 (Mal.)
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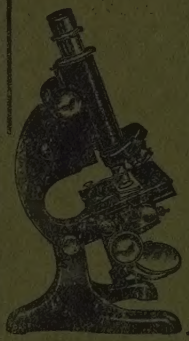

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